

EDITOR-IN-CHIEF

ANTHONY F DEPALMA
Philadelphia Pa

ASSOCIATE EDITORS

ROBERT T McELVENNY
Chicago Illinois

CHARLES W GOFF
Hartford Connecticut

EARL D MCBRIDE
Oklahoma City Oklahoma

DUNCAN C MCKEEVER
Houston Texas

DANA M STREET
Memphis Tennessee

BOARD OF ADVISORY EDITORS

ALBERT B FERGUSON SR
Brookline Massachusetts

RALPH K GHORMLEY
Rochester Minnesota

EDWARD C REIFENSTEIN JR
Bloomfield New Jersey

JAMES E M THOMSON
Lincoln Nebraska

H WINNETT ORR
Lincoln Nebraska

HARRISON L McLAUGHLIN
New York New York

Clinical Orthopaedics

ANTHONY F DePALMA
Editor-in-Chief

With the Assistance of the
ASSOCIATE EDITORS and the
BOARD OF ADVISORY EDITORS



Number Three



J B LIPPINCOTT COMPANY
Philadelphia
London
Montreal

4 13 61

COPYRIGHT 1954
BY J B LIPPINCOTT COMPANY

THIS BOOK IS FULLY PROTECTED BY COPYRIGHT
AND WITH THE EXCEPTION OF BRIEF EXCERPTS
FOR REVIEW NO PART OF IT MAY BE REPRODUCED
IN ANY FORM WITHOUT THE WRITTEN PERMIS-
SION OF THE PUBLISHERS

Clinical Orthopaedics is designed for the publication of original articles offering significant contributions to the advancement of surgical knowledge.

Original typed manuscripts not carbon copies, illustrations, should be forwarded prepaid, to Anthony F. DePalma 1025 Walnut Street, Philadelphia 7 Pa.

Manuscripts should be typed double-spaced on one side of standard typewriter paper leaving wide margins. While every effort will be made to guard against loss, it is advised that authors retain copies of manuscripts submitted. All pages should be numbered. Dorland's *American Illustrated Medical Dictionary* (edition 22) and Webster's *New International Dictionary* (edition 2) should be used as standard references. Scientific names for drugs should be used when possible. Copyright or trade names of drugs should be capitalized. Units of measurement, e.g., dosage should be expressed in the metric system. Temperature should be expressed in degrees centigrade. A contribution in foreign language when accepted will be translated and published in English.

Black and white illustrations will be reproduced free of charge but the publisher reserves the right to establish a reasonable limit upon the number. Colored illustrations ordinarily cannot be published except at the author's expense. Black and white photographs should be in the form of glossy prints. Line and wash drawings should be on white art board with lettering, in black India ink large enough to be readable after necessary reduction. Large or bulky illustrations should be accompanied by smaller glossy reproductions of the same to facilitate their circulation among the members of the editorial board. Illustrations should be numbered the tops indicated, and the author's name and the title of the article in brief should appear on the back. A separate typewritten sheet of legends for the illustrations should be supplied.

A bibliography of numbered references in alphabetical order should appear at the end of the manuscript with corresponding numbering in the text. Bibliographies should conform to the style of the *Quarterly Cumulative Index Medicus* author's name, title of article name of journal, volume number inclusive page numbers, and year of publication in the order named.

Following are the general subjects of forthcoming
issues of *Clinical Orthopaedics*

Joint Fractures and Dislocations Fall 1954

Backache Spring 1955

Present Day Status of Endoprostheses Fall 1955

Tumors of Bone Spring 1956

Chronic Hereditary Diseases and Developmental Anomalies Fall 1956

All contributors desiring to submit articles for consideration for publication on the topics listed above or in the general sections of this publication should submit them to the editor some months in advance of the date of the issue for which they are intended.

Contents

SECTION I SOFT-TISSUE CONDITIONS AROUND JOINTS

- 1 A REVIEW OF THE SURGICAL TREATMENT OF CONGENITAL DISLOCATION, RECURRENT DISLOCATION OR SLIPPING PATELLA
H Winnett Orr M D
- 2 SPRAINS
Garrett Pipkin M.D
Brief Regional Analyses
Conclusion
- 3 INJURIES TO THE LIGAMENTS OF THE KNEE
T B Quigley, M.D
- 4 CYSTS OF THE SEMILUNAR CARTILAGES OF THE KNEE
Louis W Breck, M.D
Introduction
Incidence and Location
Etiology
Pathology
Symptoms
Physical Examination
Differential Diagnosis
Treatment, After-Care and Prognosis
Report of a Series of 18 Cases of Cysts of the Menisci
Discussion
- 5 TRAUMATIC LESIONS OF MENISCI
R. A Murray M.D
Anatomy and Pathology
Symptomatology and Physical Findings
Differential Diagnosis
Conservative Treatment
Surgical Treatment
Prognosis and Complications
- 6 INJURIES OF THE MEDIAL MENISCUS
Frederick Lee Liebolt, M.D
- 7 RECURRENT DISLOCATION OF THE PATELLA
Duncan C McKeever M D
Treatment

8	TREATMENT OF DISLOCATION OF THE PATELLA	61
	Fritz Teal, M.D.	
	Etiology	61
	Mechanism of Dislocation of Patella	61
	Complications	61
	Treatment of Simple Patellar Dislocation	61
	Surgical Treatment	62
	Author's Method	
9	AVULSION OF THE HAMSTRING TENDONS FROM THE ISCHIAL TUBEROSITY	
	REPORT OF CASE	66
	Lewis M Overton, M.D., and Robert England M.D.	
	Case Report	66
	Comment	68
10	BICIPITAL TENOSYNOVITIS	69
	Anthony F DePalma M D and Gerald E Callery M D	
	Incidence	70
	Pathology	70
	Etiology	73
	Anatomic and Functional Factors	73
	Anomalies of the Bicipital Groove	76
	Degenerative Processes	77
	Clinical Features	77
	Bicipital Tenosynovitis in the Young	77
	Bicipital Tenosynovitis After the Age of 30 Years	78
	Treatment	79
	Operative Procedures	81
	Postoperative Management	81
	Analysis of Cases Reviewed	81
	Analysis of Poor Results	84
	Conclusions	85
11	RECURRENT ANTERIOR DISLOCATION OF THE SHOULDER JOINT	86
	John J Gartland, M D and John J Dowling M D	
	Treatment	87
	Operative Procedure	88
	Analysis of Results	90
12	RUPTURES OF THE ROTATOR CUFF	92
	Julius S Neviaser M D	
	Rupture of the Rotator Cuff Without Dislocation or Fracture	92
	Ruptures of the Rotator Cuff Following a Dislocation of the Shoulder	96
	Ruptures of the Rotator Cuff Following a Dislocation of the Shoulder and	
	Fracture of the Greater Tuberosity	96
	Ruptures of the Biceps Tendon	97

13 INJURIES OF THE LIGAMENTOUS AND ASSOCIATED STRUCTURES ABOUT THE HIP JOINT

T H Vinke, M D., and F M Deuschle

Anatomy of the Hip Joint
 Relations of the Hip Joint
 Movements of the Hip Joint
 Development of the Hip Joint
 Clinical Aspects

14 SPRAINS AND RUPTURES OF LIGAMENTS OF THE ANKLE JOINT

Judson D Wilson, M D

Surgical Anatomy
 Physical Examination
 The Treatment of Sprains of the Ankle
 Ligamentous Rupture with Disruption of Ankle Mortise
 Complications as the Result of Maltreatment

15 THE PELLEGRINI-STIEDA PARA-ARTICULAR CALCIFICATION

I Williams Nachlas M D

Etiology
 Pathology
 Clinical Picture
 Treatment

SECTION II

GENERAL ORTHOPAEDICS

16 GIANT-CELL TUMOR OF BONE AND ITS DIFFERENTIAL DIAGNOSIS

William C Herrick, M D and Henry L Kazal M D

Introduction
 History
 Etiology and Pathogenesis
 Clinical History and Physical Findings
 Age
 Sex
 Location
 Roentgenographic Findings
 Gross Pathology
 Microscopic Findings
 Grade I
 Grade II
 Grade III
 Differential Diagnosis
 Aneurysmal Bone Cyst
 Nonossifying Fibroma of Bone
 Ossifying Fibromas
 Chondromyxoid Fibroma of Bone
 Benign Chondroblastoma of Bone
 Unicameral Bone Cyst

16	GIANT-CELL TUMOR OF BONE AND ITS DIFFERENTIAL DIAGNOSIS (<i>Continued</i>)	
	The Biopsy	141
	Prognosis	142
17	STEP CUT KNEE FUSION WITH MEDULLARY FIXATION	145
	Wm Minor Deyerle M D, and Virgil R May Jr M D	
	Operative Technic	146
	Case Report	149
18	CLOSED MEDULLARY PINNING OF COLLES FRACTURE	152
	Leslie V Rush M D	
	Indications	153
	Time of Operation	154
	Reconstruction	155
	Stability of Fixation	158
	Anatomic Results	158
	Dynamic Factors	158
	Technical Errors and Complications	160
	Return to Function	161
	Removing the Pin	161
	Discussion	
19	A COMPARATIVE CLINICAL STUDY OF AUTOGENOUS AND FROZEN HOMOGENOUS BONE IN GRAFTING PROCEDURES	163
	Ernest A Brav Colonel, MC U.S.A	
	Statistical Data	165
	Discussion	169
20	THE USE OF TUBADIL (REPOSITORY INJECTION OF TUBOCURARINE) IN ACUTE BACK STRAIN	172
	Commander John S Thiemeyer Jr (MC) USN and Lieutenant E. F Reed Jr (MC) USN	
	Report of Cases Illustrative of the Above Therapy	173
21	FATIGUE FRACTURE OF THE SHAFT OF THE FEMUR—REPORT OF A CASE	177
	Hyman R. Osheroff Colonel MC U.S.A and Thomas C Devlin First Lieutenant, MC U.S.A	
	Case Report	177
22	VASCULAR RELATIONS OF THE ANKLE AND THEIR CLINICAL SIGNIFICANCE	179
	H. Kelikian M D	
	Arterial Pathways	179
	Summary	182
	Return Circulation	183
	Clinical Correlation	185
	Summary and Implications	208
23	TREATMENT OF FEMORAL NECK FRACTURES	209
	Richard P. Gilbert M D	
	INDEX	217

SECTION I

SOFT-TISSUE CONDITIONS AROUND JOINTS

A Review of the Surgical Treatment of Congenital Dislocation, Recurrent Dislocation or Slipping Patella

H WINNETT ORR, M.D *

There are two reasons for reviewing, for all orthopedic surgeons, the management of congenital dislocation of the patella (1) So many operative procedures have continued in use for example, release of the patella on the outer side, and reefing of the capsule on the inner side which long ago were shown to be inefficient but continue to be employed as procedures for the relief of this disability (2) In published reports of the operative care of slipping patella, it is the exception rather than the rule to find that all of the "slipping" parts involved have been controlled adequately in the postoperative care so that there was a real expectation of sound healing in correct anatomic and functional position.

The protection of the usually relaxed knee and all of its surrounding structures against stretching and strain after operation and when weight-bearing on the limb is resumed, is important not only for the prevention of the motion and the irritation that sometimes lead to postoperative wound infection but also to secure sound healing with all the parts in the relationship that will cure the disability and give "maximum benefit" for function. A proper appreciation of all this demands what Sir Robert Jones called "the orthopaedic conscience." For such care the

compression bandage, or even compression by an elastic knee "stocking" is inadequate. This is a matter of special importance in those cases in which effusion is present. When there is fluid in the knee the operation, aspiration, in addition to immobilization, is almost always

The obvious requirements of treatment in cases of slipping patella, or recurrent dislocation, have been the replacement of quadriceps tendon and its bony attachment between the femoral condyles at the knee level, the restoration of quadriceps strength and the prevention of a return of the deformity. These indications have been usually only in part, by physiologic exercises and massage by such an author as Thomas percussion of the femoral condyle to cause a hypertrophy of the articular margin of the tendon groove by mechanical devices such as pads or splints or by surgical operation. Usually the last has been shifting about of the patella and the ligament parts or the reconstruction of the mechanism of flexion and extension of leg by the rearrangement of the joint at the front of the knee. It is to these technical procedures that we shall direct particular attention.

It is of interest in this connection, to note that early American surgeons made important contributions toward straightening

*Lincoln, Neb

tabilizing deformed knees. Some years ago at a meeting of the American Medical Association in Philadelphia the Mayor not a surgeon in his address of welcome mentioned ones Physick Dorsey, Mutter Barton Gibson and others whose osteotomies and other corrections of deformity were "firsts" in laying the foundations of orthopedic surgery both at home and abroad.

That these men got training and inspiration from John Hunter the Bells, Cooper Velpeau Bichat, Larrey and Dupuytren does not detract from their achievements and the influence of their teachers at the University of Pennsylvania, and their courage as pioneer surgeons immediately after the American Revolution.

Most of the surgical expedients proposed to retain the patella between the femoral condyles have been based upon using the soft tissues in the neighborhood either to pull the patella inward or to transfer bands of tissue to the outer side to prevent the patella from following its tendency toward outward displacement. None of these has been more than partially successful, yet they have continued to be employed. Now better and sounder techniques have been proposed and will be described.

Barton,¹ in the 1830's and Gibson and Buck^{2,3} in the 1840's gave us some useful suggestions by their use of arthroplasty and osteotomy to relieve deformity and to stabilize lower extremities in good position. One might do well in many cases of knee operation, to follow their rules especially those of Mutter in the selection of patients for the treatment of fractures and the after-care of this class of patients.

Pearson a British surgeon,⁴ employed a very good, early "knee cage" brace to hold a slipping patella in correct position. However like its successors it probably was not a great success. Some of the early operators who employed incision along the outer side of the patella and "reefing" along the inner side were Canton,⁵ Roux (see Goldthwait) and Appel.⁷ The last had collected 30 cases from the literature.

William Anderson⁸ reported the treatment of a fractured patella by a technic not utilized as it should have been by others until a long time afterward. He transfixed the upper and the lower parts of the fractured bone with long pins then brought the fragments into correct relationship and fastened the pins at one end in a cork and at the other ends by wires so that all the parts were controlled in correct position. This was as good a method of pin fixation as any—except plaster of Paris—and much better than sutures wires or even the Malgaigne clamps.

Pollard⁹ released the patella on the outer side and reefed the capsule on the inner side. When this failed, he persevered with an osteotomy of the femur to get a better line for the knee later he deepened the groove for the quadriceps and resutured the capsule on the inner side, pulling the patella inward once more. Finally he got a satisfactory result. J. S. McLaren¹⁰ followed a similar program using silk sutures for the internal capsule at the last operation after 4 years he got some improvement in the knee and the limb.

In the meantime some more courageous operators J. W. Perkins,¹¹ Goldthwait¹² and Krogus¹³ introduced operative suggestions that have led to the more successful techniques and results of the present time. Perkins (1893) had a patient with a dislocation of the patella of 6 years standing. He transplanted a long and fairly wide strip of the internal capsule and the tendon over the remainder of the tendon and the patella and to the outer side of the front of the knee. In this way he carried the patella with its tendon through the open knee joint to the inner side. By suturing these structures in their new position he closed the joint with a very strong correction of the dislocation. He said that he obtained a good result. Hauser in his paper in 1938 gave Perkins somewhat less than enough credit by referring to this operation as "an attempt to free the patella and hold it in medial displacement."

In 1900 Goldthwait operated upon a patient, doing a transference of a portion of the quadriceps tendon across from the outer to the inner side in one knee and an inward transference of the tibial tubercle with its quadriceps attachment on the other. Dr Goldthwait got a better result with his tendon transference, but he expressed a preference for the bone fixation on the inner side of the tibia as being the sounder surgical procedure. Several subsequent writers, including Hauser, who used a similar transference of the tibial insertion of the tendon overlooked Goldthwait's second procedure, giving him credit for the tendon transference only.

Krogius really followed Perkins in his employment of a heavy band which he carried over from the inner to the outer side of the patella but without entering the knee joint. He sewed the band from the inner side into an incision on the outer side in front of the knee pulling the patella inward as he closed the space from which he had taken his flap. This procedure appeared, in his drawing, to disturb the mechanism of knee flexion and extension more than the more radical Perkins operation.

In connection with this discussion of knee surgery we may do well to call attention to the encyclopedic paper of Paul Heimrich¹⁴ in which the entire field is covered, but with the common difficulty that one travels long distances and arrives nowhere in particular. It provides all kinds of surgeons with all kinds of expedients but a choice of an operation for any particular occasion is difficult.

We had some experience with a problem very similar to the patellar displacement during World War I when Hey Groves at Bristol¹⁵ and S. Alwyn Smith,¹⁶ my Chief at the Welsh Metropolitan War Hospital, at Cardiff were doing their reconstructions of knee. They employed long strands of fascia through drill holes in the femoral condyles and the tibia to restore stability to some very unstable knees. There were several very satisfactory results. As I remember I

may have had something to do with the that Alwyn Smith immobilized the last of his 7 cases in plaster of Paris and secured better results than he had in his first 2. Some of the lessons we learned from these cases were not taken advantage of as prominently and as thoroughly as they should have been. A. H. Edwards¹⁷ for example, reported cases in 1920 in which he was still trying to hold slipping patellae in place with caps sutures only.

Soutter¹⁸ of Boston reported the use of a "new" fixation to the inner side of the tibia by the transference of a free fascia flap to make a reinforcement of the internal lateral ligament and capsule of the knee. He looped this strip of fascia lata through holes in the inner edge of the patella and sutured the place somewhat as Sir Robert Jones called their "tenodesis" operation. It appeared, however, that if the new ligament were tightened it interfered with flexion of the knee and increased the displacement of the patella. Soutter referred to the Goldthwait operation of 25 years before as a "transference of the outer half of quadriceps tendon which seemed to be adequate for a Boston neighbor only a few squares away."

H. Page Mauck¹⁹ proposed a new operative procedure in which he did the Goldthwait transference of the tibial tubercle with its quadriceps attachment, carried along a good part of the internal lateral ligament inward and downward to place the patella well toward the inner side of the knee and tighten all of the structures involved in the "looseness" of the patella and its tendon. All of his 5 cases were improved.

Two years later Emil D. W. Hauser²⁰ of Chicago employed a procedure which seemed to combine all the best features of the various operations described above. Hauser took out a good-sized bone block with the tibial tubercle and the quadriceps attachment transferred it inward and downward on the tibia to an opening prepared for it before hand. He was careful to get the patella in

correct alignment and to obtain the tension upon the quadriceps tendon exactly right for the stabilization of the knee joint and the range of motion of the knee. He added a reef in the internal lateral capsule to assist in correcting the valgus that is present so often.

Regarding this operation of 15 years ago, Dr. Hauser has just sent me the following supplementary comments. The quadriceps tendon, the patella and the patellar ligament are all brought to the mid-line thus re-establishing the normal pull of the quadriceps muscle from its origin to its insertion. The new insertion of the patellar ligament is lower than the original bringing about normal tension and increased strength to the quadriceps muscle.

"The countertransplant of the bone block is readily executed and gives an immediate firm attachment. The procedure is relatively simple and logical and has been successful. Dr. Hauser makes this observation:

"I feel that the Soutter ligament operation is more complicated than need be. Also it is not complete enough in that it does not presuppose a complete transplant of the quadriceps tendon and thus does not assure that the force exerted at the upper margin of the patella, where the greatest force is exerted in weight bearing when the knee is flexed, would be in the right direction."

Steindler²¹ in his second book on *Operative Orthopedics* commended the Krogius operation but for his own work added the moving of the tibial tubercle with its quadriceps attachment. He said that Horwitz had failed in 2 cases (of 20) because of inadequate postoperative physiotherapy. (There might have been insufficient support for the weak knee after the operation. See my remarks elsewhere on that point.) (H W O.)

Willis Campbell²² who borrowed the title of his book from Steindler's first edition reviews with approbation the operations of Ober (which resembled the Goldthwait tendon transference operation) Krogius and Goldthwait's tibial tubercle transfer. He also

endorsed the Hauser bone block transference of the quadriceps attachment to the inner side of the tibia. Campbell went on, as so many writers do to recommend the Gallie procedure (resembling Soutter's), elevation of the outer rim of the intercondylar groove (with which he credited Brackett, who did the operation two years before Albee) and Albee's own bone graft procedure. This fault of suggesting so many procedures, some of which must be combined with others for a good result, makes for a difficult choice by any except the most experienced surgeons. Campbell, with his own large experience and good judgment, was definite upon one matter: he employed plaster-of-Paris casts, good splints and adequate after-care to obtain real "end results" in these cases.

To recapitulate: usually too little attention has been paid to minimizing damage during and after operation even by surgeons who are supposed to have an "orthopedic conscience." Injudicious manipulations or incisions sometimes contribute to disability instead of repair.

The old injunction to "do the patient no harm" should be borne in mind, especially at the operating table. After-care is if anything even more important. Valgus knees, backward displacement of the tibia on the femur and undue stretching of the quadriceps tendon and the knee ligaments are all factors that have been neglected in the surgery of recurring dislocation of the patella. It always should be borne in mind that replacement of the patella in correct position is only one of the items in a reconstruction of the anatomy and the physiology of the affected knee joint.

I cannot close without reminding some of you that in our own work I have not hesitated to employ pin fixation through both tibia and femur and in plaster-of-Paris casts until healing is well established. The patient is certainly safer and more comfortable with this kind of immobilization.

For several years past, Dr. Fritz Teal and our other associates have performed Patel-

Bibliography

- lectomies in cases of recurrent dislocation of the patella Degenerative changes have been found on the adjacent articular surfaces of the patella and the femoral condyles At the time of the operation these changes were sufficiently severe to suggest that the more conservative procedures would be inadequate The results have been good One large teen age girl had the patellae removed on both sides and got an excellent result Duncan McKeever (in a letter) suggests his patelloplasty in such cases

REFERENCES

- 1 Barton John Rhea A new treatment in a case of ankylosis of the knee Am J M Sc 21 320 1837
- 2 Gibson William Operation for ankylosis of the knee Am. J M Sc 4 39 1842
- 3 Buck Gordon Resection of the knee, Am J M Sc 10-277 1845
- 4 Pearson Pad for recurrent dislocation of the patella Lancet 2 12 1884
- 5 Canton Operation for recurrent dislocation of the patella Lancet, March 24 1860
- 6 Roux On slipping patella. See Goldthwait.
- 7 Appel Report of 30 cases of congenital and recurrent displacement of the patella, Munchen. med. Wchnschr June 25 1895
- 8 Anderson William Lancet October 1892
- 9 Pollard Report of a case in which he an osteotomy of the femur Lancet M 30 1891
- 10 McLaren J S Ann Surg. 31 679 1901
- 11 Perkins, John W Complete dislocation of the patella reduced by arthrotomy af years Ann Surg 18 654 1893
- 12 Goldthwait J E. J Am Orthop September 1895
- 13 Krogus Centralbl f Chir 31 254 1905
- 14 Heinrich, Paul Surg., Gynec & Obst 177 1909
- 15 Groves E. W Hey Lancet 2 674 1917
- 16 Smith S A Brit J Surg 6 176 1918
- 17 Edwards A H Brit. J Surg. 8 20 1920
- 18 Soutter J A M.A 82 1261 1924
- 19 Mauck, H P J Bone & Joint Surg. 984 1936
- 20 Hauser E D W Surg. Gynec & C 66 199 1938
- 21 Steindler A Orthopedic Operations, 448 Springfield Ill. Thomas, 1940
22. Campbell, Willis Operative Orthopedics p 451 St. Louis Mosby 1939

BIBLIOGRAPHY

- Bradford E. H Slipping patella Boston M 134 1896
Hoffa Habitual luxation of the patella Arch klin Chir 59 543 1899

*See the separate communication by Dr Teal in this volume

Ruptures of the Rotator Cuff

JULIUS S. NEVIASER, M.D. *

Although the entity "Ruptures of the Rotator Cuff" has gained widespread acceptance since the publication of Codman's text, *The Shoulder* the diagnosis and the management remain beclouded with the aura of confusion, half truths and misunderstanding. In order to diagnose and treat this entity correctly one must have a complete knowledge of the anatomy, the physiology and the pathology of the musculotendinous cuff. The cuff consists essentially of the tendons of the supraspinatus, the infraspinatus, the teres minor and the subscapularis which fuse with the articular capsule of the shoulder joint, forming a continuous envelope encasing the head of the humerus. The first three tendons insert in their respective facets on the greater tuberosity and the latter tendon on the lesser tuberosity of the humerus.

It must be borne in mind that although the supraspinatus is most prone to be involved in a rupture it is quite possible to have a severe injury of this tendon without significant impairment of total shoulder function. Formerly the erroneous concept was prevalent that derangement of supraspinatus function materially impaired shoulder activity specifically initiating and maintaining abduction. The fallacy in this concept may be demonstrated dramatically by abolishing the protective pain reflex by the injection of a local anesthetic agent and observe the patient performing the act of abduction and maintaining this abduction without difficulty.

The primary function of the supraspinatus is fixation of the humeral head in the glenoid. However, if this tendon is ruptured, the deltoid may elevate the humerus. This is accompanied by some motion of the scapula and a disturbance in the normal glenohumeral rhythm. If this is remembered, plus understanding that abduction can also be gained by the remaining intact cuff elements by humeral rotation, the diagnosis and the treatment of these lesions assume a simple and logical pattern.

Ruptures of one or more components of the rotator cuff usually occur in one of three ways:

1 There may be a rupture following a history of injury without a dislocation or a fracture.

2 A rupture may occur following a dislocation of the shoulder.

3 Rupture of the rotator cuff may follow a dislocation of the shoulder with a fracture of the greater tuberosity of the humerus.

RUPTURE OF THE ROTATOR CUFF WITHOUT DISLOCATION OR FRACTURE

This lesion usually occurs through degenerated tendon tissue. Therefore it most frequently occurs not earlier than the fourth or fifth decade of life. Whether the rupture of the supraspinatus tendon is partial or complete, it always will occur at the so-called "critical area" near the insertion of the tendon into the bony tuberosity of the humerus (Fig. 1). This so-called "critical



FIG. 3 Incomplete avulsion fracture of the greater tuberosity which may be associated with a partial rupture of the supraspinatus tendon.

or more the likelihood is that the patient has a small tear and requires conservative treatment rather than surgery. Injection of procaine into the suspected area of injury removes the protective muscle spasm and pain and enables the patient to abduct the arm if there are only minor tears. A complete inability to initiate or maintain abduction in any of the humeral arcs usually indicates a massive cuff tear involving all of the cuff insertion. Ten cc. of 1 per cent procaine is generally sufficient for a test. If the procaine injection does not allow the patient to abduct his arm to at least 90° or if he has difficulty maintaining abduction against resistance then arthrography may be performed. From 6 to 8 cc. of radiopaque material is injected under fluoroscopic control at the anterior portion of the shoulder joint. The injection is made about 1 inch below the acromioclavicular joint. If the fluid runs out immediately into the subdeltoid bursa, one may assume that the patient has a substantial tear and that surgery is indicated.

There is an occasional lesion in which we find a partial tear involving the deep layer of cuff fibers near their greater tuberosity insertion with incomplete avulsion of the tuberosity. These cases present the same physical findings as the incomplete ruptures, but the roentgenograms will show the incomplete tuberosity fracture (Fig. 3). They respond quite well to conservative treatment.

In the treatment of partial ruptures we can anticipate a very satisfactory result in most instances by conservative means. The small or incomplete tears may heal, or the short rotator muscles of the shoulder may compensate for the loss of action of the injured tendon by a change of the rotation arc. A sling may be all that is needed during the acute painful stages. Graduated exercises are then allowed, and most patients will regain their full range of motion in a period ranging from 3 to 6 weeks. When one is convinced that the patient has a complete tear as determined by clinical examination as well as by procaine injection and arthrography then surgery can be done to approximate the edges of the torn tendon cuff. There may be times when one is in doubt as to whether or not surgery is indicated, and a wait of 10 days or 2 weeks will not jeopardize the ultimate result. By this time the reaction from injury and the pain will have subsided so that one can better evaluate the extent of the patient's injury. If the patient still cannot abduct the arm to 90° after this period and if secondary changes appear such as atrophy of the infraspinatus and the supraspinatus muscles, making the scapula spine prominent, then one is justified in exploring these cases. In operating upon these patients I prefer the transacromial approach as advocated by McLaughlin. When the lesion has been exposed all suturing should be done to restore normal anatomic position of the structures but without any tension. Reinsertion of the retracted portion of the musculotendinous cuff into the humeral head is done at whatever point it will reach without tension and also with the arm at the side. In some cases after exposure one may have difficulty in

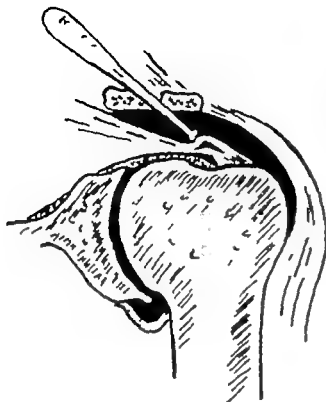


FIG. 4 Confirmation of a partial tear by buckling of the tendon with an instrument.

locating the site of pathology. This is particularly true of incomplete ruptures or partial tears through the cuff.

There are some valuable points which will be helpful in locating these lesions at the operating table. Normally when the arm is abducted the cuff slides under the coracoacromial arch of the shoulder smoothly with hardly any wrinkle on the tendon surface. However at the site of tear the tendon may become elevated into a blister just before it passes under the edge of the acromion. One can confirm the presence of this tear also by passing the flat surface of an instrument laterally toward the tuberosity along the tendon surface. If this buckles, then this is the site of cuff tear (Fig. 4). When the tear has been located then it can be repaired in the usual manner. In massive ruptures of the shoulder the cuff may be turned in between the head of the humerus and the glenoid (Fig. 5). If so an attempt should be made to repair this lesion without tension even if it is necessary to fix part of the cuff edges into the superior surface of the head (Fig. 6). However if the cuff is greatly re-



FIG. 5 Massive ruptures may show complete separation of the entire cuff, which may be turned in between the glenoid and the humeral head.



FIG. 6 Reinsertion of the retracted cuff as far medially as is necessary to secure fixation without tension.

tracted and the sutures seem to be under tension, then a primary fusion of the shoulder joint is possibly the best method of repair. My own experience has shown that these massive ruptures with marked separation and retraction of the cuff do not do well by operative repair. The sutures tend to pull out when motion is allowed. Then again, it must be remembered that massive



FIG 7 Note upward riding of humeral head, following reduction of dislocation



FIG 8 Dislocation with fractured tuberosity fragment opposite the glenoid.

ruptures occur in the older age group where soft tissues are degenerated and the bone is osteoporotic

RUPTURES OF THE ROTATOR CUFF FOLLOWING A DISLOCATION OF THE SHOULDER

This is a common injury. The torn cuff may be the only disabling factor following such an injury. One can easily understand why this is so. When the head of the humerus is pulled out of position without any damage to the greater tuberosity one must suspect a tear of the musculotendinous cuff. After reduction of the dislocation and immobilization of the arm the patient can best be tested after an interval of 10 days or 2 weeks. If he does not show any evidence of abduction after the acute phase has subsided then operative repair is indicated. Roentgen examination is most important in the diagnosis of ruptures associated with dislocations or fractures. Inasmuch as massive ruptures are likely to occur in this type of injury, roentgenograms may show such a lesion following a reduction by the upward riding of the humeral head (Fig. 7). This is due to lack of support of the overlying structures as the tear has extended through

the full thickness of the tendon and the joint capsule and possibly the floor of the bursa. Then again, with the patient standing, a subluxation of the head of the humerus may be demonstrated by roentgenograms taken with the arm hanging or holding a heavy weight. An attempt should be made to repair this type of lesion without tension and if this is not possible a fusion of the shoulder joint would be indicated.

There are certain instances of dislocations when after reduction a persistent subluxation of the humeral head may occur and if this is so it generally indicates a large cuff tear. This is usually seen in elderly people where the degenerative changes are most pronounced. If such is the case and the patient has a very definite disability as determined by the tests previously described, then operative repair is in order.

RUPTURES OF THE ROTATOR CUFF FOLLOWING A DISLOCATION OF THE SHOULDER AND FRACTURE OF THE GREATER TUBEROSITY

Dislocation of the shoulder with a fracture of the greater tuberosity nearly always is accompanied by a torn cuff. Although this may be our most frequent injury so far as cuff tears are concerned many such tears do not require operative repair. However some may result in serious disability if they are not managed properly. The roentgen



FIG. 9 Dislocation with tuberosity fragment retracted up under the acromion



FIG. 10 Dislocation with the tuberosity fragment following the humeral head

films give us an idea of what we are dealing with right from the beginning. If the greater tuberosity fragment remains in its normal position opposite the glenoid (Fig. 8) one usually has a tear of the aponeurosis between the lesser and the greater tuberosity allowing the humeral head to dislocate. Usually this dislocation is intracapsular. When the dislocation is reduced the edges of the longitudinal tear generally heal well with good function. Fortunately this is the most common occurrence in fracture dislocations of the shoulder. If the tuberosity fragment should be retracted up under the acromion (Fig. 9) then one must presume a large and extensive tear exists. In addition, the tuberosity fragment under the acromion acts as a wedge between the humerus and the acromion blocking glenohumeral motion. Early operative repair should be done through the transacromial approach, and restoration of the tuberosity fragment to the shaft of the humerus can be maintained by sutures or screws. I prefer the screw as this allows the patient to start early motion resulting in a shorter postoperative period of disability. If the tuberosity fragment ac-

companies the humerus (Fig. 10) it should make one very suspicious of a cuff tear. After reduction the patient should be watched closely and if there is no return of abduction power after 10 days or 2 weeks then surgery is indicated for repair of the cuff tear.

RUPTURES OF THE BICEPS TENDON

The long tendon of the biceps at its intra-articular portion is intimately connected to the musculotendinous cuff of the shoulder. Because degenerative changes also take place in this tendon it is easy to understand why it is possible to have a rupture of the biceps tendon at this area. Also one may have a rupture of the biceps tendon associated with a rupture of the rotator cuff of the shoulder. Then again, a massive tear involving the rotator cuff with retraction of the tendon of the subscapularis medially and the external rotators laterally may cause the tendon of the biceps to be displaced anteriorly over the lesser tuberosity. Inspection of the bicipital groove will reveal the tendinous tunnel to be empty. Clinically the diagnosis of a

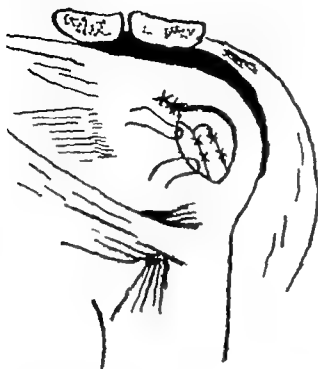


FIG 11 Repair of ruptured biceps tendon by fixation in the upper portion of the bicipital groove

rupture of the biceps is relatively easy. In addition to the previously mentioned signs of cuff tear one may have a prominent muscle belly which is quite noticeable in the lower portion of the arm. In the treatment of these ruptures the tendon is best replaced by insertion into the bicipital groove fixed close to the upper portion of this groove.

Afterward, the ruptured cuff can be repaired (Fig 11)

SUMMARY

The various types of rotator cuff injuries of the shoulder have been reviewed. The diagnosis and the treatment as presented should be regarded as a logical sequence to an understanding of the fundamentals of anatomy and physiology of the structures about the musculotendinous cuff. Conservative treatment suffices in most cases; however, surgical repair of the cuff is indicated in those few instances that present a definite disability after the initial traumatic reaction has subsided, which usually is from 10 to 14 days after injury.

BIBLIOGRAPHY

- Codman, E. A. *The Shoulder* Boston, Thomas Todd, 1934
- McLaughlin, Harrison L. Lesions of the musculotendinous cuff of the shoulder *J Bone & Joint Surg* 27:31 1944
- Muscular and Tendonous Defects at the Shoulder and Their Repair *Lectures on Reconstruction Surgery* pp 343-358, Ann Arbor Edwards, 1944
- Moseley H. P. *Shoulder Lesions*, ed. 2, New York, Hoeber 1953
- Neviaser Julius S. Common injuries of the musculotendinous cuff of the shoulder *Bull. Hosp Joint Dis.* 14:58 1953

Injuries of the Ligamentous and Associated Structures about the Hip Joint

T. H. VINKE, M.D. * AND
F. M. DEUSCHLE†

This paper is written with the purpose of bringing together several of the clinical entities involved in injuries to the hip joint. Since injury to the hip joint produces changes in the anatomy in and about the joint, we feel that much is to be gained from a critical review of the anatomy of the joint and the surrounding structures as well as a presentation of several new interpretations of these structures in the light of function in health and injury. Much of the following material amounts to a review but several new concepts are considered sufficiently important to be incorporated into the broad thesis of this presentation.

ANATOMY OF THE HIP JOINT

The hip joint is a ball-and-socket joint composed of the head of the femur and the acetabulum of the hip bone. The rim of the acetabulum is completed by the transverse acetabular ligament, and the fossa is deepened by the glenoid labrum. Union is maintained by a capsular ligament which is reinforced by the ischiofemoral, the pubofemoral and the iliofemoral ligaments.

The transverse acetabular ligament bridges the acetabular notch and creates the acetab-

ular foramen for nerves and blood vessels entering the joint.

The lips of the glenoid labrum slope so that the mouth of the acetabulum is narrowed to grip the head of the femur more securely.

In front of the joint the capsular ligament is especially strong. It is attached behind and above to the transverse ligament and below and in front to the labrum. The femoral attachments are to the trochanteric line in front, the upper and the lower surfaces of the neck near the trochanter and to the neck behind, $\frac{1}{2}$ inch from the intertrochanteric crest. Longitudinal and circular fibers are present in the capsule. The circular fibers encircle the neck of the femur to form a collar—*zona orbicularis*. The circular fibers are the deeper fibers except at the back of the neck of the femur where they appear superficially. The stronger and more numerous fibers are longitudinal and are most numerous at the surface of the capsule where they are collected into the named ligaments. Some of these fibers at the femoral attachment are reflected onto the neck of the femur as the retinacula, which are frequently strong enough, especially in the young, to prevent displacement of the neck of the femur when fractured.

The iliofemoral, the ischiofemoral and the pubofemoral ligaments are divisions of the sheet of the superficial longitudinal fibers.

* Assistant Clinical Professor of Orthopedic Surgery, College of Medicine, University of Cincinnati.

† Assistant Professor Applied Anatomy, College of Medicine, University of Cincinnati.

The iliofemoral is in front of the capsule, appearing as an inverted "Y" because the part between the limbs is thinner than the sides. The apex of the ligament is attached to the anterior inferior iliac spine and the base to the trochanteric line. The apical attachment is especially strong, and frequently the ligament resists tearing so well that the antero-inferior spine of the ilium is fractured.

The pubofemoral and the ischiofemoral ligaments pass obliquely from the pubic and ischial acetabular rims to the femoral attachments, mingling with the circular fibers of the capsule.

The ligamentum teres is attached at its apex to the fovea capitis of the femur and at its base by three bands: one to the transverse acetabular ligament and a second and a third to the pubic and the ischial margins of the acetabular notch respectively.

The cavity of the joint is lined by a synovial membrane. It covers the two surfaces of the labrum and continues over the acetabular fat body and the ligamentum teres enclosing its neck in a sheath. It is reflected from the femoral attachment onto the neck up to the articular margin of the head. The sheath of synovial membrane on the neck is raised into folds: the *retinacula*, by blood vessels running to the head. Tearing of these in fracture of the neck diminishes the blood supply to the head. Sometimes a bursa under the psoas may communicate with the joint cavity through an opening between the iliofemoral and the pubofemoral ligaments.

The blood supply to the hip joint must be divided into two sets: vessels running to the capsule and others to the head. The capsule is supplied constantly by the superior gluteal, the inferior gluteal, the medial and the lateral femoral circumflex arteries and not infrequently by the deep femoral artery and the capsular branches of the internal pudendal artery. The acetabular artery supplies the transverse ligament, adjoining parts of the capsule and part of the acetabulum. Supplemental blood supply of the acetabulum is gained from the gluteals and the obturator. Recently Weathersby¹ reported that the fo-

veal artery to the ligamentum teres came from the obturator in 54 of 104 hips from stillborn infants from the medial circumflex femoral in 14 and from both arteries in 6 specimens. In 30 other hips the arteries joined before the origin of the acetabular branch.

The medial and the lateral circumflex femoral give the branches to the head of the femur found in the *retinacula*. In 20 of 49 femora of the Weathersby series the foveal artery penetrated the head. There was anastomosis between the capital and the foveal arteries in only 3. This is consistent with the findings of Wolcott² and others³ and supplements their work.

The nerves to the hip joint are derived from the nerves to the rectus femoris: the anterior division of the obturator (sometimes the posterior too) and the nerve to the quadratus femoris. It is to be noted that the obturator and the femoral nerves innervate both the hip and the knee joints and also that the nerve to the quadratus femoris is connected to 3 (4 5 L 1 S) of the 5 spinal nerves forming the sciatic (4 5 L, 1 2 3 S) which innervates the knee joint indirectly. Thus, pain referred from one joint to the other is explained.

RELATIONS OF THE HIP JOINT

The *gluteus minimus* covers the superior surface of the capsule. Inferiorly the obturator externus approximates the lower surface of the capsule and a portion of the back of the neck of the femur beyond the capsule. After the medial circumflex femoral artery has passed between the pectineus and the psoas, it lies below the joint, then ascends along the obturator externus to its insertion. The insertion of the *iliopsoas tendon* is below the lower part of the capsule of the joint.

Three muscles lie anterior to the capsule. From medial to lateral, these muscles are the pectineus, the psoas and the *iliacus*. A few fibers of the *iliacus* are inserted into the capsule. A bursa separates the psoas from the capsule. Lateral to the *iliacus* is the rectus femoris. Its straight head covers the

upper end of the iliofemoral ligament, while its reflected head is attached to the capsule beneath the gluteus minimus. The iliofemoral ligament at its distal lateral attachment of the femur lies between the rectus femoris and the gluteus minimus, which are covered by the tensor fasciae latae and separated from it by fatty connective tissue containing the ascending branches of the lateral femoral circumflex artery. The deep edge of the tensor fascia latae is attached to the capsule.

Posteriorly from above downward, are the piriformis, the obturator internus and the gemelli and the upper edge of the quadratus femoris. The nerve and the vessels to the latter muscle descend between the combined obturator internus and gemelli and the bone and then between the quadratus femoris and the capsule.

MOVEMENTS OF THE HIP JOINT

Flexion is produced by the rectus femoris, the sartorius, the pectineus, the obturator externus, the adductor longus and brevis, the iliopsoas, the pubic part of the adductor magnus and the upper anterior portion of the gluteus medius and minimus. Flexion is inhibited by contact of the neck of the femur with the acetabular margin, by contact of the thigh and the abdomen when the knee is flexed and by the hamstrings when the knee is extended.

Extension is produced by the gluteus maximus, the hamstrings and the ischial portion of the adductor magnus. Extension is limited by the iliofemoral ligament.

Abduction is brought about by the gluteus medius and minimus, the upper anterior fibers of the gluteus maximus, the tensor fasciae latae and the sartorius and in flexion by the piriformis and the obturator internus. The pubofemoral ligament limits abduction.

Adduction is accomplished by the adductors, the pectineus, the gracilis, the quadratus femoris and the inferior portion of the gluteus maximus. Adduction is limited by the ischiofemoral ligament and by contact with the other thigh. If the hip is flexed,

the lateral part of the iliofemoral ligament limits adduction.

Lateral Rotation is obtained by the gluteus maximus, both obturators posterior, fibers of the gluteus medius and minimus, both gemelli, the piriformis, the quadratus femoris, the sartorius, the pectineus, the adductor longus, brevis and magnus and, in flexion, the psoas.

Medial Rotation is gained by the anterior parts of the gluteus medius and minimus and the tensor fasciae latae and, during flexion, by the iliopsoas.

Greatest laxity of the joint occurs when the joint is flexed, adducted and rotated slightly medially. This is the position frequently taken by the joint when effusion into the joint occurs.

DEVELOPMENT OF THE HIP JOINT

In the fetus of the eighth week the three cartilaginous elements—ilium, ischium and pubis—meet at the Y acetabular suture. The pubic element is the last to chondrify. In the ninth week a synovial cavity appears, cartilaginous outgrowths occur from all three of the above elements especially the iliac, and a head is separated from the shaft of the femur by the development of a neck. The synovial lining of the joint results from an ingrowth of cells from the periphery into the blastema between the head of the femur and the hip bone. The intra articular ligament, the ligamentum teres, appears in the fetus in the capsule of the joint. The isolation of the ligamentum teres in the joint results from a development of the head expanding as a wing on each side of the ligamentum teres, the fusion of which separates the ligament from the capsule. The reflected part of the ligament on the inferior surface of the neck is the part of the capsule with which the ligamentum teres is continuous.

Congenital dislocation of the hip may result from arrest of development of the acetabular elements of the ilium, the ischium and the pubis.

The hip joint is complete early in the third month.

CLINICAL ASPECTS

The hip joint apparently is not involved in serious ligamentous difficulties as frequently as are joints such as the knee and the ankle. The study of these ligamentous structures may aid in understanding certain phases of hip pathology and its treatment. Much has been written about fractures involving the neck of the femur, as well as prosthetic appliances and reconstructive procedures.

It may be assumed that the hip is not involved frequently in sprains and ruptures of its various ligaments. However the fact is that frequently the ligamentous structures about the hip joint are injured. We have all seen many cases of children who have developed a lump of unknown origin. We are pressed as to what is the cause of such a limp. The laboratory findings, including blood counts, sedimentation rates, roentgenograms, etc., all may be negative. There is no evidence of infection or allergic manifestation. In many of such cases by close questioning we find that the child has placed unusual strain on his hip either by accident or by some strenuous athletic or acrobatic pursuit. On examination, we find a few tender areas and certain motions producing or aggravating pain. Such cases in the younger age groups are relieved of symptoms by restricted activities for a few days and sometimes a few weeks. Older patients may require crutches or a longer period of restricted activity as far as the hip joint is concerned. These strains usually are produced either by wide abduction or by wide adduction. In wide abduction the pubofemoral ligament is strained and in wide adduction the ischiofemoral ligament is strained (Figs 1 and 2). Consequently the symptoms should be aggravated by the motion which produces the strain. Marked flexion does not produce strain, since it is inhibited by the contact of the neck of the femur with the acetabular margin and by the contact of

the thigh against the anterior abdominal wall. However by violent extension or exercise the iliofemoral ligament can be strained.

The hip joint is a weight-bearing joint and it is controlled by some of the strongest ligaments in the body. Dislocation of the hip joint is quite rare because the hip joint is held firmly in the acetabulum. The motion of the hip joint is universal because of its ball-and-socket arrangement. Sprains are less common in this joint because of this architectural design, associated with strong ligamentous structures. A dislocation of this joint produces extensive soft tissue damage. The ligamentous structures are ruptured, and the synovial membrane is torn badly. Occasionally it is associated with fractures of the acetabulum and damage to the head of the femur. The extent of damage is at times difficult to determine. Of course in extensive soft tissue damage, there is interference with the blood supply and subsequent aseptic necrosis of the head of the femur. Prolonged periods of no weight-bearing and restricted activity become quite necessary. For other than prompt reduction there is very little else one can do to prevent an aseptic necrosis.

In dislocations the medial circumflex artery is compressed easily against the tendon of the obturator externus by the head of the femur. This is especially true in a high dislocation, i.e., a dislocation above the obturator externus. It is obviously important that this compression against the medial circumflex artery be released as soon as possible in order to re-establish the blood supply.

The usual mechanism of the traumatic dislocation of the hip is the result of a strong force exerted against the knee when the hip and the knee are flexed. This occurs frequently in automobile accidents when the hip joint is flexed and adducted, for the surrounding ligaments are lax and then the head of the femur can be dislocated with ease over the posterior rim of the acetabulum. By far the greatest number of dislocations of the hip are posterior while anterior obturator and central dislocations are also possible. In a posterior dislocation of the

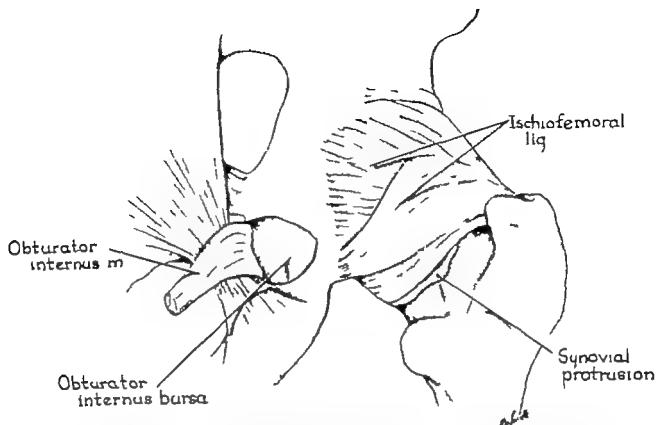


FIG. 1 Attention is called to the relation of the ischiofemoral ligament in the capsule. This relation shows why this is taut in adduction. We point out the structure of the bursa between the tendon of the obturator internus and the ischial tuberosity. Since this is an unique cartilage-tendon joint unrecognized pathologic changes resulting from injury to the obturator internus tendon should be considered in diagnosis and treatment.

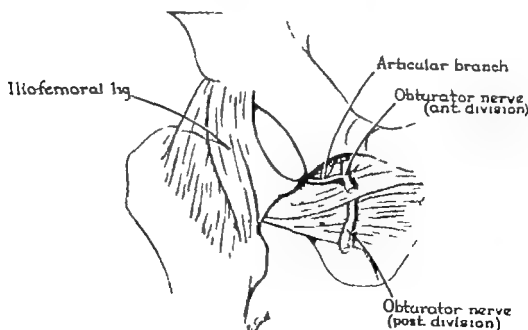


FIG. 2. Observe the relationship of the obturator nerve, both anterior and posterior divisions, to the obturator externus muscle.

hip the involved extremity is held in internal rotation and adduction combined with moderate flexion, and there is obvious shortening of the involved lower extremity. The greater trochanter appears to be quite prominent. With the patient recumbent, the knee is rotated inwardly and flexed. The foot is inverted, and the heel often rests on the dorsum of the opposite foot. The head of the femur can be felt indistinctly in its posterior position through the gluteal muscles, while the trochanter lies anteriorly and above Nelaton's line. According to Steindler's posterior dislocation of the hip is held in a position of inward rotation and adduction, which is the result of the fulcrum action of the iliofemoral ligament which winds itself around the neck of the femur in such a way as to aid in holding the femur in internal rotation.

The anterior dislocation is more infrequent, and in such a dislocation the leverage action occurs at the greater trochanter and at the posterior aspect of the neck of the femur. The head of the femur is forced against the rim of the acetabulum, the latter acting as a fulcrum over which the head is brought out through a rent in the anterior and inner portion of the capsule. In an anterior dislocation the head of the femur is held in outward rotation. If the head is rotated inward from this position, lever action at the fulcrum of the greater trochanter will lead it downward toward the obturator foramen. If inward rotation should continue around the acetabulum, a posterior dislocation could be produced. The rents and tears in the capsule of the hip joint apparently heal quite rapidly hence little strain is placed on them once the dislocation is reduced. In the anterior obturator dislocations, the extremity is held in external rotation and abduction, the knee being flexed. The shortening is not very apparent. Sometimes there may be slight lengthening. The head may lie in the obturator foramen. The external rotation and eversion of the foot are less marked, while the abduction is increased. When the head lies in the higher

pubic position, there is less abduction but more external rotation. In both cases, there is moderate flexion of the hip as well. It is impossible to extend or adduct the limb. In the high position the head can be felt in front of the pubis, but when it is in the obturator foramen, it is difficult to locate. The greater trochanter is absent from its usual position, being displaced inward, and a depression remains instead. Occasionally there is pain and numbness in the distribution of the femoral nerve when the head of the femur is in the obturator foramen because the femoral nerve is compressed between the taut iliacus and psoas muscles.

The decisions for definitive treatment of such injuries about the hip must be based on several factors: (1) the general condition of the patient, (2) an appraisal of the injured hip and (3) thorough knowledge of the involved anatomy. Roentgenograms always should be taken to determine whether or not a fracture or a dislocation is present. In many cases a fracture dislocation occurs, the rim of the acetabulum being fractured. This injury usually is accompanied by considerable shock. The amount of soft-tissue damage that is present will influence the rate of repair. In severe dislocations and sprains of the hip there is definite soft tissue damage, repair of which may be delayed because of the lack of circulation. Restoration of circulation can be facilitated by elevation of the extremity by early muscular activity and by physiotherapy consisting of heat and massage. However weight-bearing should not be permitted until enough time has elapsed for complete healing of the soft tissues. Various amounts of time for restricted weight bearing have been suggested. After a fracture dislocation it is our opinion that the patient should be immobilized in a plaster hip spica cast for approximately 4 to 6 weeks. Others advise a period of immobilization of from 4 to 8 weeks. After this, exercises should be instituted gradually and the patient should be on crutches without weight bearing for another 6 weeks to allow sufficient time for healing of the ligamentous

and the vascular structures. If incomplete healing is suggested by pain and muscle spasm after the 3 months have elapsed further restriction of weight bearing should be carried out. Many advise that a period of 6 months of no weight bearing is the best treatment.⁵ However it seems reasonable that those cases which appear to have normal function without pain can be allowed to bear weight after 3 months have elapsed. Immediately after a dislocation of the hip emergency traction should be applied with the least possible manipulation and with no attempt at reduction. If the general condition permits reduction under a general anesthesia should be done as soon as possible. The longer the head of the femur remains out of the acetabulum the greater the soft tissue damage. Early reduction of the dislocation under a complete anesthesia and gentle maneuvers causes less damage to the soft tissues and circulation than when great force or violent manipulation is used.

All methods of reduction begin with flexion of the hip. Many methods of reduction of the hip have been described. It is noted that a circumduction maneuver of the hip and the knee would replace most hips. If reduction of the head of the femur must be delayed for any reason, one can apply skeletal traction holding the hip and the knee flexed at about 90°. Sometimes this alone will reduce the dislocation. The best manipulative reduction for both posterior and anterior dislocations is to flex the knee and the hip into position and rotate the lower extremity into its neutral position so that the femoral head lies below the acetabulum then, by traction the head is pulled or elevated gently into the acetabulum. Forceful manipulation is very seldom necessary. Open reduction of dislocations of the hip should be the last resort of treatment, since open reduction increases the amount of damage to the blood supply. If there is a fracture of the rim of the acetabulum such a fracture also may be reduced at the time of dislocation. If the fracture of the posterior rim of the acetabulum is large enough to produce

instability of the hip open reduction must be done and the fractured portion held in position with one or more screws. The number of screws depends on the size of the fragment of bone being replaced.

Watson Jones⁶ has stated that aseptic necrosis develops in about 30 per cent of traumatic dislocations of the hip. In uncomplicated cases the patient should be kept in bed several weeks until the soft tissues of the hip have a chance to heal. After that, the patient should be instructed to use crutches for at least 3 months, and if no abnormal roentgen findings are present and when normal function is established weight-bearing may be permitted. Roentgenograms should be taken of the hip every 2 or 3 months for at least 12 months to determine whether or not the head is becoming necrotic. The only method we have to prevent disability after a dislocation of the hip is to reduce it immediately and to protect the hip from weight bearing until the blood supply has been restored. However according to the statistics of Vernon Thompson,⁷ the length of time before full weight bearing was resumed in his cases did not significantly affect the occurrence of aseptic necrosis; he found that the causes of this avascular necrosis depend upon the extent of damage and thrombosis to the blood vessels supplying the head of the femur.

It should be mentioned that paralytic or infectious lesions which cause flexion-adduction contractures or spasm may give rise to a spontaneous dislocation of the hip because in this position the femur is unprotected by the acetabulum and it lies almost entirely in contact with the joint capsule.

The sciatic nerve may be traumatized in posterior dislocation due to stretching of the nerve over the head of the femur or occasionally to compression of the nerve in the sciatic notch. This produces a paralysis of the muscles of the posterior thigh and those below the knee and a wide area of anesthesia.

Incomplete nerve lesions recover if the pressure is relieved by immediate reduction of the dislocation. But if there has been

considerable delay, the prognosis of recovery of nerve function becomes poor.

Dislocations of the hip may damage the periosteum, the muscle attachments and the capsule. Subperiosteal hematoma may occur and subsequently undergo calcification. Such a hematoma absorbs more rapidly if the hip is reduced immediately and the periosteum is replaced in its normal position. After delayed reduction or after rough manipulations a greater hematoma develops and as a result large areas of calcification or a traumatic myositis ossificans may occur.

Redislocation of the hip may occur particularly if fracture of the posterior margin of the acetabulum is present. In such cases plaster cast immobilization for at least 6 weeks is preferable. It also might be stated that early motion in such cases of extensive capsular damage increases the danger of thrombosis of the vessels and of subsequent aseptic necrosis of the head of the femur. During the period of immobilization in plaster casts a course of muscle setting exercises including quadriceps exercises, is very valuable and should be carried out during the entire period of immobilization.

At times it appears on roentgenograms that a hip dislocation has been reduced in the anteroposterior projection, but in reality it still is dislocated completely. In such cases the femoral head has been pulled down to the level of the acetabulum. There appears to be a fairly good position of the hip except for the fact that Shenton's line is not continuous and does not form an arch suggesting that the proper anatomic alignment has not been restored. In such cases it is also important to note that one is unable to see the lesser trochanter because of the position of the femur in the anteroposterior view: the hip has been rotated internally so much that the head of the femur is behind the acetabulum. Of course lateral roentgenograms of the hip in addition to the anteroposterior views would definitely show whether or not the hip has been reduced.

In cases of old unreduced dislocations, surgical intervention is definitely indicated.

If the hip has been dislocated for many weeks or months manipulative reduction is not possible. Even by surgery reduction becomes extremely difficult in such old cases and it usually is complicated by an aseptic necrosis of the head of the femur. In posterior dislocations the posterior surgical approach, such as that described by Gibson, is preferable. Usually in such cases either an arthrodesis or a prosthesis becomes necessary. The cup arthroplasty as described by Smith Petersen is still the choice of many orthopedic surgeons. In anterior dislocations of the hip in addition to the torn capsule, the adductor muscles and the obturator externus usually are lacerated. The Smith-Petersen incision is indicated in the anterior dislocation if surgery is performed in the posterior dislocation, the short external rotators, particularly the obturator internus muscles are liable to injury. The tendon of the obturator internus may become interposed between the neck and the acetabulum and interfere with reduction.

In all dislocations of the hip the following tissues are damaged or macerated and often are associated with extensive hematoma: the synovial membrane, the capsule and the associated ligaments, particularly the iliofemoral, the ischiofemoral and the pubofemoral and the ligamentum teres. The iliofemoral ligament is so strong that it rarely is ruptured completely. The blood supply of the capsule and the head of the femur is disrupted, since the retinacula of the synovial membrane contain blood vessels running to the head of the femur. Branches of the following blood vessels also are involved, since they supply the capsule: the superior gluteal, the inferior gluteal, the medial and the lateral femoral circumflex and often the deep femoral and capsular branches of the internal pudendal artery. The foveal artery is severed completely since it is associated with the ligamentum teres. If the transverse ligament is damaged the acetabular artery may be involved.

Nerves derived from branches of the anterior division of the obturator and the nerve

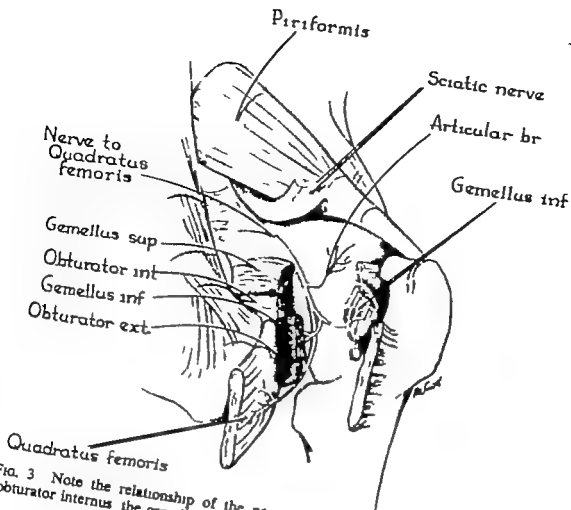


FIG. 3 Note the relationship of the nerve to the quadratus femoris to the obturator internus the gemelli the quadratus femoris and to the joint capsule

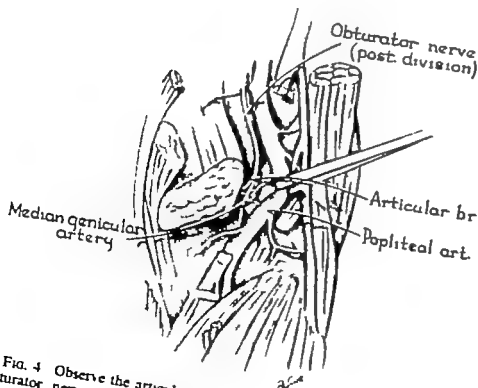


FIG. 4 Observe the articular branch of the posterior division of the obturator nerve accompanying the median genicular vessels to the knee joint.

to the quadratus femoris are damaged in dislocations of the hip and, as noted elsewhere in this paper the obturator and the femoral nerves innervate both the knee and the hip joints (Figs 3 4 and 5) and the nerve to the quadratus femoris is connected to 3 of the 5 spinal nerves forming the sciatic. Pain about the knee in cases of hip pathology is produced because of this anatomic arrangement. This innervation of the hip joint makes it difficult to obtain complete anesthesia of the hip joint.

We have attempted to consolidate the anatomic and clinical relationships of the ligamentous and associated structures about the hip joint. Consideration has been given to several hip problems, such as traumatic dislocations. We believe that ligamentous strains involving this joint are frequent and important. Treatment designed to prevent aseptic necrosis has been discussed.

REFERENCES

- 1 Weathersby Hal T The arterial supply of the hip joint, *Abstr Anat. Rec.* 115 378, 1953
- 2 Wolcott, W Eugene *Surg., Gynec. & Obst.* 77 61 1943
- 3 Trueta, J., and Harrison, M. H. M. *J Bone & Joint Surg.* 35B.442, 1953
- 4 Steindler *Mechanics of Normal and Pathological Locomotion in Man* Baltimore, Charles C. Thomas, 1935
- 5 Stuck and Vaughn *South. Surgeon* 15 659 1949
- 6 Watson Jones *Fractures and Joint Injuries*, Baltimore, Williams & Wilkins 1946
- 7 Thompson Vernon P and Epstein, Herman C. *J Bone & Joint Surg.* 33A 746 1951

Note The authors are grateful to Dr Roger C. Crafts and Dr Joseph A. Freiberg for reading the manuscript and giving several valuable suggestions.

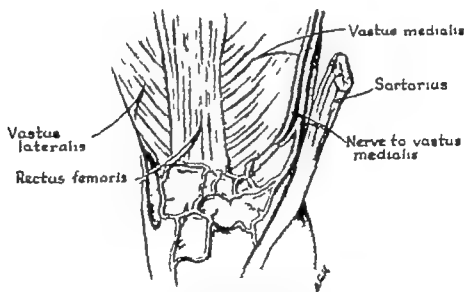


FIG 5 The nerve to the vastus medialis, a branch of the femoral, gives articular branches to the knee joint.

Sprains and Ruptures of Ligaments of the Ankle Joint

JUDSON D WILSON, M.D *

A sprain of the ankle is due to a sudden inversion or eversion strain produced by stepping on an uneven surface or by a fall. The body weight is suddenly thrust on the ligaments of the ankle which, at the moment, is unprotected by supporting muscles and tendons surrounding the ankle joint. With a moderate strain, the ligamentous fibers are only stretched, or perhaps a few of them are torn. In a simple sprain, the stability of the ankle joint is not disturbed. However if the sprain is severe a complete avulsion or tearing of the ligamentous fibers occurs and this permits undue mobility and a temporary dislocation of the talus. This is particularly true when there is a complete avulsion of the anterior and the middle fasciculi of the lateral ligament. A temporary dislocation of the ankle joint is recognized only by special clinical and roentgenologic tests.

SURGICAL ANATOMY

In discussing sprains and ruptures of the ankle joint, it is imperative to review briefly the anatomy and the physiology of this joint. The lower end of the tibia expands and becomes four sided. The medial portion extends distalward, forming the medial malleolus. The lateral surface presents the fibular notch and a rough depression for the attachment of the inferior interosseous ligament of the distal tibiofibular joint. The

lower extremity of the fibula forms the lateral malleolus which extends approximately 1 cm. lower than the medial malleolus. The joint is in the form of a mortise and tenon, the two malleoli forming the clasping surface of the mortise. The articular surface of the tibia is concave and articulates with the superior surface of the talus, which is wedge shaped, with the base in front. The stability of the joint of the foot and the ankle depend a great deal on the accurate approximation of the talus to the mortise formed by the tibia and the fibula. The talus consists of a body which articulates with the tibia and the fibula by its upper or trochlear surface a forward projecting neck and a head which articulates with the navicular. The inferior surface of the body of the talus rests on the calcaneus, which is able to rotate slightly beneath it. Because of this inversion and eversion take place at these joints and the ankle joint is spared much wear and tear. However these movements are not sufficient to protect the ankle from severe and sudden strains. The superior surface of the talus which is arched evenly anteroposteriorly rocks in the tibiofibular notch and is broader anteriorly.

The tibia and the fibula are connected by a strong interosseous ligament (Fig 1) which is the inferior prolongation of the interosseous membrane by the inferior transverse ligament which extends from the posterior part of the medial surface of the

lateral malleolus to the posterior margin of the distal extremity of the tibia and by the antero-inferior tibiofibular ligament.

The capsular ligament of the ankle is relatively weak, particularly anteriorly and posteriorly. The anterior ligament is a thin membrane attached to the anterior aspect of the malleoli and the lower extremity of the tibia, and inferiorly to the superior surface of the neck of the talus (Fig 2). The posterior ligament is the weakest of the ankle ligaments and is represented by a few ligamentous bands connecting the posterior surface of the tibia and the posterior tibiofibular ligament to the posterior aspect of the talus below (Fig. 3).

The ligaments which reinforce the medial and the lateral aspects of the joint, are very strong and pass fanlike from the malleoli to the tarsal bones. The internal lateral (deltoid) ligament strengthens the medial side of the joint to a great degree (Fig 2). The proximal extremity of the ligament attaches to the anterior medial malleolus. The distal portion presents an unbroken line of attach-

ment to the navicular, the talus and the posterior calcaneonavicular ligament. It is reinforced by the tendons of the tibialis posterior and the flexor digitorum longus.

The lateral ligament is weaker and less complete. It is divided into three components: the anterior, the middle and the posterior fasciculi (Fig 1). The anterior talo-fibular ligament extends from the anterior border of the lateral malleolus to the lateral surface of the neck of the talus. The calcaneofibular ligament, the middle ligament, is a band extending obliquely downward and backward from the malleolus to the lateral surface of the calcaneus. The posterior fibulo-calcaneal ligament binds the fibula to the talus and is rarely torn.

The patient presents himself complaining of severe pain and swelling on the outer surface of the foot, resulting from a turning of his ankle or a fall. If the sprain is a severe one the swelling and the ecchymosis become pronounced at a rapid rate, as a result of an extravasation of blood into the soft tissues. After a few hours the entire

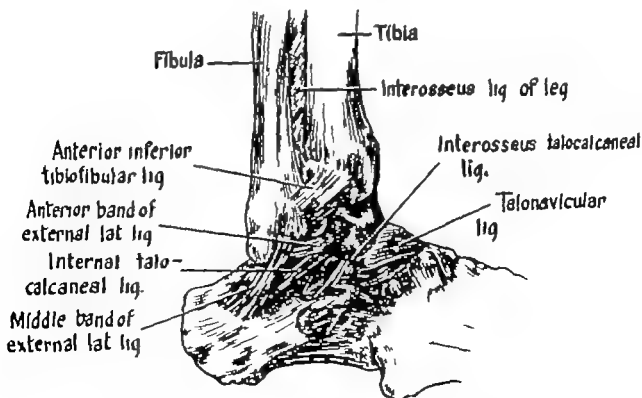


FIG. 1 Ligaments of external or lateral surface of ankle joint.

foot and lower leg may become edematous and discolored. If the sprain is a mild one the patient may be able to bear partial weight on the foot whereas, if it is a severe one, he may or may not be able to bear any weight. It is characteristic of sprain that the pain persists in spite of immobilization and placing the extremity at rest.

PHYSICAL EXAMINATION

On physical examination, when the injured ankle is seen within an hour or two after injury there may be little objective evidence of severe injury. During the first few hours, a very well-circumscribed swelling forms a semicircle around the tip of the fibula with tenderness at the point of the origin of the lateral ligament. In other cases there is an egg-shaped, localized swelling largely below the tip of the fibula, becoming subsequently more diffused and more marked. Tenderness and swelling on the medial aspect of the ankle over the deltoid ligament is an important sign. In a complete rupture of the lateral ligament, there is a

temporary dislocation of the talus and a resultant injury to the deltoid ligament. The history of something snapping, giving way or slipping out of place is significant, and a lateral ligament rupture should be suspected. Tenderness of the distal tibiofibular joint, indicating some damage to these ligaments is a constant finding. The absence of deltoid ligament trauma, that is, no swelling or tenderness on the medial side of the ankle joint, is indicative of a simple sprain. In such a sprain one can be reasonably sure that there is no fracture, in that all motions of the foot are possible even though painful, especially on inversion of the foot.

The more severe sprain may simulate a fracture. Consequently, meticulous x ray examination is important in every case. Three preliminary views of the ankle should be insisted upon with every ankle injury: an anteroposterior view wherein the malleoli are equal distance from the x ray films; a true lateral view and an oblique view with the foot and leg rotated internally at 45°. If there is any suspicion of a spreading of the

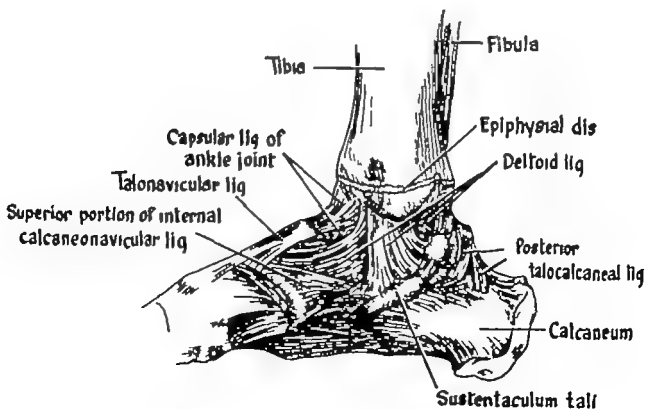


FIG. 2. Ligaments of internal or medial surface of ankle joint.

ankle mortise or a tear of the two components of the external lateral ligament, an x ray examination should be made with inversion stressed using either local or general anesthesia. When the roentgenogram is taken, the foot is forcibly inverted by grasping the forefoot with one hand and applying counterpressure against the middle third of the leg with the other hand while the knee is maintained in a true anteroposterior plane. The same maneuver is repeated on the uninjured side in order to determine the normal amount of mobility of the talus.

Leonard reported a study of 51 cases of inversion injury of the ankle wherein 23 patients were shown roentgenographically to have had spontaneously reduced subluxation. Both ankles were x rayed in inversion, and the difference in degree of parallelism between the articular surface of the talus and the tibia of the two sides was considered as diagnostic of a spontaneously reduced subluxation of the ankle. In order to visualize the mechanism of inversion injury resulting

in ligamentous damage and to ascertain the importance of a given ligament in a maintenance of talotibial stability experiments were carried out on the cadaver to determine the course of the ligaments in varying degrees of flexion and dorsiflexion and of the effect of cutting the ligament in various combinations to determine ankle stability. He concluded that the anterior talofibular ligament was the most frequently ruptured and the most important component of the lateral ligament of the ankle. He concluded further that inversion films should be taken with the foot in equinus if the tenderness is anterior to the fibula and at 90° if the tenderness is posterior to the fibula.

THE TREATMENT OF SPRAINS OF THE ANKLE

The treatment depends upon the severity of the sprain and the amount of swelling present. In a mild or simple sprain, immediate treatment can be instituted. The area,

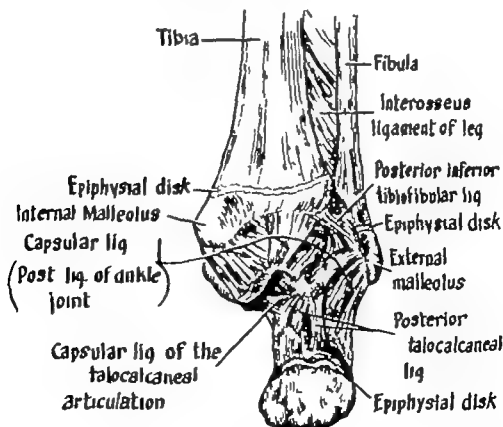


FIG. 3 Ligaments of posterior surface of ankle joint.



FIG 4 Method of applying adhesive strips for basket weave splint.



FIG 5 End result of basket weave adhesive splint for simple sprain.

including the lower third of the leg the ankle and the foot is cleansed thoroughly and shaved. Then the swollen, tender area is injected with 5 to 10 cc of a 2 per cent solution of procaine and the area is massaged lightly to ensure infiltration of the procaine. Next the prepared area is then painted with Mercurochrome tincture of benzoin or merthiolate. It has been our experience that 5 per cent Mercurochrome has been more effective in the prevention of skin irritations than either the tincture of benzoin or the merthiolate.

Then the patient is seated on the examining table and the foot is supported in a position of 90° dorsiflexion and eversion. This can be accomplished by a 1 inch band of adhesive tape around the plantar surface of the foot at the base of the toes beneath the metatarsal heads. The patient is instructed to hold the adhesive tape in such a way that the desired dorsiflexion and eversion of the foot is maintained. Adhesive

rolls, 1 and 2 inches wide should be at hand. The rolls of adhesive make application easier and the desired length can be obtained without waste. The adhesive strips are applied as follows: use the 1 inch strip and begin on the inner aspect of the leg at the level of the middle and the lower thirds. Secure the adhesive by exerting slight pressure with the left hand and passing down over the plantar surface of the heel and up the outer side of the leg. The second strip is started on the inner side of the foot at the level of the first metatarsal head, passed around the heel over the first strip and along the outer border of the foot, parallel with the plantar surface and ends at the level of the fifth metatarsal head. Strip No 3 is placed slightly anterior and parallel with strip No 1 (Fig. 4). The strapping is continued in the following manner: Bring vertical strips forward and horizontal strips upward on the foot and the leg. Upon reaching the anterior surface of the leg the vertical

strips are continued in their normal course by curving around the leg. At this stage the dorsum of the foot and the surface of the anterior and posterior leg is covered with short strips of the 2 inch adhesive. In this manner the entire foot and the lower leg are covered with a basket weave type of splint (Fig. 5). It is important that the position of dorsiflexion and eversion be maintained throughout the strapping.

The foot and the leg are then bandaged with a gauze bandage for 12 to 24 hours to ensure adherence of the tape to the skin. The patient may be permitted full weight-bearing on the involved foot. It is well to have the outer border of the heel and the sole elevated 3/16 inch to maintain eversion of the foot and to relieve the strain on the injured ligament. The adhesive will loosen in 7 to 10 days due to perspiration, natural oils of the skin and motion of the foot. The adhesive splint is then removed, the skin is cleansed thoroughly with some solvent such as carbon tetrachloride and the foot is re-strapped in the manner described before. The adhesive strapping and the shoe elevation should be maintained until healing is complete. The average sprained ankle in the adult requires from 4 to 6 weeks to heal in children, it heals more quickly.

In the severe sprain, where the entire foot and the lower leg are swollen, discolored and excruciatingly painful, the treatment

is more complicated. A very satisfactory method of treatment is to cleanse the foot and the leg thoroughly and inject the area of maximum swelling with the enzyme, hyaluronidase using 2 per cent procaine as a vehicle.

Investigation by Seifert at the Wyeth Institute demonstrates that hyaluronidase acts directly on the ground substance of certain body tissues, freeing hyaluronic acid. This acid forms the main constituent of the normal body fluid. With a breakdown of the natural barriers there is a freer flow of the intercellular fluid. It has been shown that hyaluronidase has no propulsive power but that by freeing the intercellular barriers, the accumulated fluid can be dispersed rapidly by the use of external pressure.

The solution is prepared by dissolving one ampule containing 1000 T.R. (turbidity reduction) units of the enzyme in 5 cc of 2 per cent procaine. This amount can be doubled if the area to be injected is extensive. By the use of a 2 per cent procaine solution, total anesthesia can be obtained locally and the necessary x ray evaluation can be made to determine the extent of the ligamentous tear.

After x ray examination, a compression dressing is applied. A very simple and efficient form of pressure dressing can be devised by wrapping the foot, the ankle and the lower leg in several layers of glazed



FIG. 6. Elastic compression bandage to reduce swelling of severely sprained ankle.



FIG 9 Roentgenogram of the ankle joint following a severe sprain that appeared to be normal in regard to joint structure.



FIG 10 Roentgenogram of the same ankle after the injection of 2 per cent procaine and forcing the foot in a lateral direction. This demonstrates a tear of the medial collateral ligament, as well as the interosseous membrane.



FIG 11 Reposition of the talus and internal fixation of the fibula with vitallium screw

LIGAMENTOUS RUPTURE WITH DISRUPTION OF ANKLE MORTISE

As previously stated, the ankle mortise consists of the distal ends of the fibula and of the tibia. The integrity of this all important mortise is maintained by the strong tibiofibular syndesmosis and the internal collateral ligament. In case of a disruption of one or both of these ligaments or a combination with a fracture of the distal end of the fibula a lateral displacement of the talus may result (Fig 7 B). Failure to correct the abnormality of this joint will result in disastrous arthritic changes in the ankle joint.

If the routine roentgenograms of the injured ankle joint disclose an incongruity between the mortise and the talus every effort must be made to effect a complete normal restoration. Normally the lateral surface of the talus is in line with the lateral body of the tibia (Fig 7 A). Early treatment of such a condition is essentially the same as that of an acute sprain except that the foot is placed very slightly in the equinus position since the normal anatomic widening of the talus may force the fibula lateralward. Roentgenograms are obtained in anteroposterior, lateral and oblique positions following the application of the cast. If a normal restoration of the ankle mortise has been restored, no further treatment is required other than immobilization until healing has taken place. If it is found that a normal restoration has not been obtained, an open reduction is indicated.

We have found that a very satisfactory method of restoring the integrity of such a joint is by open reduction and internal fixation with a special vitallium screw. The screw that is used (Bosworth type) has a wide, thin head and very coarse threads on the distal one third (Fig 8). The intervening space is smaller in diameter than the distal end and has a smooth surface. The patient is anesthetized, the cast is removed, a pneumatic tourniquet is applied, and then the foot is cleansed surgically and draped sterilely. A short incision is made over the anterolateral



FIG 12. A fracture dislocation of the ankle with diastasis of the fibula and a fracture of the internal malleolus.

aspect of the distal end of the fibula, exposing the distal end of this bone. The space between the tibia and the fibula is inspected and freed from any intervening soft tissue or blood clot. This allows an easy replacement of the displaced fibula. A drill hole a little larger than the proximal end of the screw is placed through the fibula extending into the tibia. A screw of the appropriate length is selected and inserted into the previously made hole. The large coarse threads on the end of the screw permit considerable traction without tearing through the cortex of the tibia (Figs 9 to 11). This will pull the fibula into its normal position and the reduction is maintained. Fracture of the distal end of the fibula associated with a diastasis may be treated in the same manner (Fig. 7 D).

If there is an avulsion of the internal malleolus (Fig 7 C) a short incision is made directly over the internal malleolus, the reduction is accomplished, and the fragment is anchored into normal position with an ordinary vitallium screw (Figs 12, 13). If the fragment of the internal malleolus is very small the entire fragment is removed, and the ligamentous structures is reattached di-



FIG. 9 Roentgenogram of the ankle joint following a severe sprain that appeared to be normal in regard to joint structure.



FIG. 10 Roentgenogram of the same ankle after the injection of 2 per cent procaine and forcing the foot in a lateral direction. This demonstrates a tear of the medial collateral ligament, as well as the interosseous membrane



FIG. 11 Reposition of the talus and internal fixation of the fibula with vitalium screw

LIGAMENTOUS RUPTURE WITH DISRUPTION OF ANKLE MORTISE

As previously stated the ankle mortise consists of the distal ends of the fibula and of the tibia. The integrity of this all important mortise is maintained by the strong tibiofibular syndesmosis and the ternal collateral ligament. In case of a disruption of one or both of these ligaments or a combination with a fracture of the distal end of the fibula, a lateral displacement of the talus may result (Fig. 7 B). Failure to correct the abnormality of this joint will result in disastrous arthritic changes in the ankle joint.

If the routine roentgenograms of the injured ankle joint disclose an incongruity between the mortise and the talus every effort must be made to effect a complete normal restoration. Normally the lateral surface of the talus is in line with the lateral body of the tibia (Fig. 7 A). Early treatment of such a condition is essentially the same as that of an acute sprain except that the foot is placed very slightly in the equinus position, since the normal anatomic widening of the talus may force the fibula lateralward. Roentgenograms are obtained in anteroposterior lateral and oblique positions following the application of the cast. If a normal restoration of the ankle mortise has been restored, no further treatment is required other than immobilization until healing has taken place. If it is found that a normal restoration has not been obtained, an open reduction is indicated.

We have found that a very satisfactory method of restoring the integrity of such a joint is by open reduction and internal fixation with a special vitallium screw. The screw that is used (Bosworth type) has a wide thin head and very coarse threads on the distal one third (Fig. 8). The intervening space is smaller in diameter than the distal end and has a smooth surface. The patient is anesthetized, the cast is removed, a pneumatic tourniquet is applied, and then the foot is cleansed surgically and draped sterily. A short incision is made over the anterolateral



Fig. 12 A fracture dislocation of the ankle with diastasis of the fibula and a fracture of the internal malleolus.

aspect of the distal end of the fibula, exposing the distal end of this bone. The space between the tibia and the fibula is inspected and freed from any intervening soft tissue or blood clot. This allows an easy replacement of the displaced fibula. A drill hole a little larger than the proximal end of the screw is placed through the fibula, extending into the tibia. A screw of the appropriate length is selected and inserted into the previously made hole. The large coarse threads on the end of the screw permit considerable traction without tearing through the cortex of the tibia (Figs 9 to 11). This will pull the fibula into its normal position and the reduction is maintained. Fracture of the distal end of the fibula associated with a diastasis may be treated in the same manner (Fig. 7 D).

If there is an avulsion of the internal malleolus (Fig. 7 C) a short incision is made directly over the internal malleolus. The reduction is accomplished and the fragment is anchored into normal position with an ordinary vitallium screw (Figs 12, 13). If the fragment of the internal malleolus is very small, the entire fragment is removed, and the ligamentous structures are reattached di-



FIG 9 Roentgenogram of the ankle joint following a severe sprain that appeared to be normal in regard to joint structure



FIG. 10 Roentgenogram of the same ankle after the injection of 2 per cent procaine and forcing the foot in a lateral direction. This demonstrates a tear of the medial collateral ligament, as well as the interosseous membrane.



FIG 11 Reposition of the talus and internal fixation of the fibula with vitalium screw

reposition of the talus beneath the tibia, and reduction could not be maintained. Upon open reduction, it was found that the proximal portion of the fibula had been displaced behind the tibia and was caught behind the posterior lateral ridge of the tibia. The fibula was held in this position by the tight stress of the interosseous membrane above. In order to reduce the displaced fibula, a pry was inserted between the tibia and the fibula, and with considerable force the fibula was pried out from behind the tibia and moved into its proper place. After the fibula was freed, reduction of the fracture dislocation was easily secured.

It was the author's opinion that the mechanism of this condition was a result of the foot twisting under the talus with the leg continuing to push forward and to rotate outward. The lateral collateral ligaments drew the intact fibula behind the tibia. Continuation of the force rotates the talus backward and out from its position beneath the tibia, causing further force on the lateral collateral ligament, finally the fibula is broken off against the posterior tibial border. There is no ligament or soft tissue remaining attached to the upper fragment of the fibula by which it can be drawn back into place. It was the author's conclusion that a proper reduction can be obtained only by open operation and prying the proximal end of the fibula into its proper location on the lateral surface of the tibia. Often, a misinterpretation of the roentgenograms is made because the roentgenologist believes that the view taken was not truly a lateral or anteroposterior exposure. In cases where one is unable to obtain an accurate reduction of the talus and the fibula, such a condition should be suspected and, if found to be true should be corrected. It is likely that some of the malpositions noted in old fractures of the ankle joint are probably due to this condition and are responsible for the traumatic arthritis which necessitates an arthrodesis of the ankle joint.

We have seen one case wherein a patient fell from a ladder and produced an upward

dislocation of the talus between the fibula and the tibia without fracturing the bones. Reduction was accomplished easily, and the fibula was transfixed with the coarse thread vitallium screw as described above. The end result was very satisfactory.

COMPLICATIONS AS THE RESULT OF MALTREATMENT

Untreated cases or failure to maintain immobilization until healing is completed in a sprained ankle may result in disaster to the ankle joint. Occasionally a maltreated simple sprain develops an acute traumatic bone atrophy or osteoporosis. Such a condition is usually referred to as a Sudeck's atrophy. He regarded it as a reflex neurotrophic phenomenon. Its progress is rapid, and there is a marked loss of calcium from the bones of the extremity, particularly in the bones distal to the injured area.

The condition is characterized by a consistent and excruciating pain. The extremity is edematous, cold and tender. At first the skin is moist, red and glossy but then takes on a dry parchmentlike texture. The joints are stiff and painful. The patient becomes very apprehensive, and usually sedation is required. Roentgenographically the bones present a characteristic moth-eaten atrophic appearance. This condition may persist over a period of several months and may resist all forms of treatment.

We have obtained the best results by placing the patient on a high-calcium diet, complemented by large doses of vitamin D and moderate doses of thyroid, provided that it is tolerated. In the very acute cases the extremity is immobilized by a compression bandage. Then the patient is placed at rest with the extremity slightly elevated and under a heat cradle. Daily physical therapy in the form of whirlpool, contrast baths and light massage is instituted. Graded exercise is instituted and encouraged but is given only as tolerated. These exercises are increased against resistance as the condition improves. Full weight bearing should not be permitted until improvement is well along.

rectly to the tibia. Then the wound is closed, sterile dressings are applied, and their positions are maintained by sterile glazed cotton. The foot is held in slight plantar flexion and eversion, and a plaster cast is applied from the toes to below the knee maintaining this position. The cast is removed at the end of 4 weeks and the foot is strapped in the usual basket weave fashion until the fractures or the ligaments have healed.

A few cases of complete dislocation of the ankle joint without an associated fracture have been reported in the literature. The only comprehensive report dealing with this type of injury is that of Wilson, Micheale and Jacobson, who collected 14 cases which had been reported since 1913 and added 2 cases of their own. In general, the mechanism of the injury was a fall upon the foot with the foot held in plantar flexion. The

sudden weight of the body on the foot in the plantar flexed position forced the talus backward into a complete dislocation without fracture. It is apparent that severe and extensive damage was done to the collateral ligament in all instances as well as the syndesmosis of the tibiofibular joint. The reduction was accomplished easily under general anesthesia. The foot and the leg were encased in a cast until the ligaments healed. The end results of these cases were reported as good.

Bosworth reported 5 cases of fracture dislocation of the ankle joint in which satisfactory reduction could not be accomplished by closed methods. Roentgenograms revealed a typical bimalleolar fracture with marked displacement of the talus rupturing the anterior ligaments of the ankle. Attempts at closed reduction revealed a poor



FIG. 13 Reduction and internal fixation with vitalium screws.

reposition of the talus beneath the tibia, and reduction could not be maintained. Upon open reduction, it was found that the proximal portion of the fibula had been displaced behind the tibia and was caught behind the posterior lateral ridge of the tibia. The fibula was held in this position by the tight stress of the interosseous membrane above. In order to reduce the displaced fibula, a pry was inserted between the tibia and the fibula, and with considerable force the fibula was pried out from behind the tibia and moved into its proper place. After the fibula was freed reduction of the fracture dislocation was easily secured.

It was the author's opinion that the mechanism of this condition was a result of the foot twisting under the talus with the leg continuing to push forward and to rotate outward. The lateral collateral ligaments drew the intact fibula behind the tibia. Continuation of the force rotates the talus backward and out from its position beneath the tibia, causing further force on the lateral collateral ligament finally the fibula is broken off against the posterior tibial border. There is no ligament or soft tissue remaining attached to the upper fragment of the fibula by which it can be drawn back into place. It was the author's conclusion that a proper reduction can be obtained only by open operation and prying the proximal end of the fibula into its proper location on the lateral surface of the tibia. Often, a misinterpretation of the roentgenograms is made because the roentgenologist believes that the view taken was not truly a lateral or anteroposterior exposure. In cases where one is unable to obtain an accurate reduction of the talus and the fibula, such a condition should be suspected and, if found to be true, should be corrected. It is likely that some of the malpositions noted in old fractures of the ankle joint are probably due to this condition and are responsible for the traumatic arthritis which necessitates an arthrodesis of the ankle joint.

We have seen one case wherein a patient fell from a ladder and produced an upward

dislocation of the talus between the fibula and the tibia without fracturing the bones. Reduction was accomplished easily and the fibula was transfixed with the coarse thread vitallium screw as described above. The end result was very satisfactory.

COMPLICATIONS AS THE RESULT OF MALTREATMENT

Untreated cases or failure to maintain immobilization until healing is completed in a sprained ankle may result in disaster to the ankle joint. Occasionally a maltreated simple sprain develops an acute traumatic bone atrophy or osteoporosis. Such a condition is usually referred to as a Sudeck's atrophy. He regarded it as a reflex neurotrophic phenomenon. Its progress is rapid, and there is a marked loss of calcium from the bones of the extremity particularly in the bones distal to the injured area.

The condition is characterized by a consistent and excruciating pain. The extremity is edematous, cold and tender. At first the skin is moist, red and glossy but then takes on a dry parchmentlike texture. The joints are stiff and painful. The patient becomes very apprehensive, and usually sedation is required. Roentgenographically the bones present a characteristic moth-eaten atrophic appearance. This condition may persist over a period of several months and may resist all forms of treatment.

We have obtained the best results by placing the patient on a high-calcium diet, complemented by large doses of vitamin D and moderate doses of thyroid provided that it is tolerated. In the very acute cases the extremity is immobilized by a compression bandage. Then the patient is placed at rest with the extremity slightly elevated and under a heat cradle. Daily physical therapy in the form of whirlpool, contrast baths and light massage is instituted. Graded exercise is instituted and encouraged but is given only as tolerated. These exercises are increased against resistance as the condition improves. Full weight-bearing should not be permitted until improvement is well along.

case fails to respond to conservative a skin type plaster cast with a walking should be applied. The patient is engaged to begin gradual weight bearing by use of crutches.

lateral sympathectomy or procaine of the lumbar sympathetic ganglia as well as deep x ray therapy have been recommended.

complete restoration of the ankle mortise will result in an enlarged, painful ankle. In such a case a traumatic arthritis will develop and complete relief can be obtained after an arthrodesis of the talotibial

adequate treatment of a ruptured external lateral ligament results in a so-called loose ankle. This condition presents an unstable ankle and is responsible for repeated sprains or recurrent dislocations. This condition is very disabling and exerts a deep psychological effect on the patient. The treatment is surgical repair of the ligament.

SUMMARY

A sprained ankle is an important injury and must be treated according to the severity of the injury. A simple sprain should be immobilized for approximately 3 weeks with an adhesive basket weave splint. Failure to do so may result in prolonged pain, chronic swelling, and edema and occasionally acute traumatic bone atrophy. Severe sprains require in addition to routine roentgenograms lateral inversion films to rule out a complete tear of the ligaments of the ankle.

A severe sprain that is complicated by marked edema and subcutaneous hemorrhage is treated by injecting the involved

area with the enzyme hyaluronidase using 2 per cent procaine as a vehicle. This is followed by a compression bandage and bed rest until the edema has subsided. The treatment then consists of immobilization, with the foot everted and dorsiflexed until the ligaments are healed.

Selected cases, wherein the ankle mortise is disturbed, are treated by immobilizing the fibula through internal fixation and the application of a plaster boot cast.

Studies on the cadaver reveal that the anterior talofibular ligament is ruptured most frequently and is the most important component of the lateral ligament.

A properly treated sprain or ruptured ligament of the ankle joint gives uniformly good results wherein as a maltreated sprain may result in disaster to the ankle joint.

BIBLIOGRAPHY

- Anson, B. J. and Maddock, W. G.: *Callander's Surgical Anatomy* ed. 3 Philadelphia Saunders, 1952.
- Wilson, N. J., McKeale, A. A., and Jacobson, E. W.: Ankle dislocations without fracture. *Bone & Joint Surg.* 21:198 1949.
- Key, J. A. and Copwell, H. E.: *Management of Fractures, Dislocations, and Sprains*, ed. 5 St. Louis, Mosby 1951.
- Leonard, Morton H.: Injuries of lateral ligaments of ankle. *Bone & Joint Surg.* 31 A:313 1949.
- Mayer, Victor: Ankle mortise injuries, *Surg. Gynec. & Obst.* 96:98 1953.
- Dunlap, Knox: Recurrent subluxations of ankle masquerading as sprains, *U.S. Armed Forces M.J.* 2:445 1951.
- MacAusland, W. R., Jr., Gartland, J. J., and Hallock, H.: The use of Hyaluronidase in orthopedic surgery. *J. Bone & Joint Surg.* 35-A:604 1953.

The Pellegrini-Stieda Para-articular Calcification

I WILLIAM NACHLAS, M.D

The discovery of the x ray in 1895 made available a new diagnostic medium that brought to light skeletal anomalies that previously had not been known. One of these anomalous structures was a calcareous shadow that was found at the knee joint medial to the condyle. In 1905 Pellegrini¹ described this finding and in 1907 Stieda² reported 5 cases. Subsequently a number of case reports of this condition appeared in the literature under the name "Pellegrini Stieda disease." More recent articles on the subject have appeared under such titles as "Shadows of the Epicondyle of the Internal Femoral Condyle,"³ "Post traumatic Para-articular Ossification of the Knee Joint,"⁴ and "Para-articular Calcification of the Knee."⁵

The common denominator in the essays on this subject is an x ray shadow overlying the lower end of the femur on its medial aspect. The shadows seen in the illustrations that have appeared in the literature are by no means uniform in shape. Nor are they always in the identical location. Furthermore the interpretations that have been offered as to the origin of the shadow the causative factors and the tissues involved have differed appreciably. Indeed, as one surveys the literature one wonders if a number of distinct pathologic conditions have not been grouped under one classification. However there is a nucleus of cases that are similar clinically and roentgenographically

so that one is justified in considering them as a clinical entity. The name that is suggested for this affection is "para articular calcification of the knee."

ETIOLOGY

This disease though not generally known cannot be considered as rare. Several writers who have been interested in this subject have been able to find and report fairly large series. Bistolfi is said to have found 78 typical cases in a series of 767 roentgenograms of injured knees, approximately 10 per cent.⁶ Finder⁷ who reviewed the traumatic lesions of the knee in the orthopedic department of Cook County Hospital found that in the course of 1½ years 42 patients, or 3 per cent, showed calcifications medial to the femoral condyle. Of these 28 resembled those described by Pellegrini and Stieda. Perocchia reported 39 cases. In an Army General Hospital of about 2 000 beds with a rather slow turnover of its population the writer found 20 typical cases in one year.⁸ Russell and Smith,⁹ working in the x ray department of a similar hospital found 8 cases in 9 218 examinations. Kulowski gives the case histories of 11 patients, and in a footnote indicates that 2 additional cases came to his attention between the time his paper was offered for publication and the date of its printing.⁴ It is not improper to assume that as the condition becomes more generally recognized and

the search for it by means of x ray examination is expanded, the lesion will be found to be quite common.

Most of the patients reported have been men. However, one must consider the fact that the reports have included studies of industrial or military groups which normally have a predominance of males. In Finder's figures, obtained in a civilian hospital, the ratio was 5 males to 2 females. Kulowski's 11 patients included 8 males and 3 females. Wetzler and Elconin⁹ reported 3 cases of which 2 were females. It is likely that the distribution will vary with exposure to trauma rather than with any inherent sex characteristics.

Young adults in the prime of life chiefly between the ages of 30 and 40 have supplied the material for most of the reports. The lesion does occur in younger people but only a few have been found in patients under 20. The oldest case reported is a patient 71 years old.

In all the studies the causative factor most generally found is trauma. Since the injury is not always the result of a major force and since several weeks may elapse between the

injury and the appearance of the x ray finding, the patient may not always recognize the causal relationship. Careful questioning will usually establish the occurrence of an antecedent trauma. Commonly the injury is of the indirect type—a twist of the knee or a lateral bend of the leg at the knee joint. However a number of patients indicate that their injury consisted of a direct blow on the inner side of the knee. Also it has been reported that the mass developed following the therapeutic manipulation of a stiff joint. Some of the affected knees have had previous inflammatory conditions. In one series it was noted that a number of the patients had had a "weak knee" as the result of earlier trauma but the lesion did not appear until a relatively minor injury had produced a new disability.⁸ The precipitating force need not be of great magnitude. One is led to conjecture on the possibility of the production of the condition by repeated minor injuries in the same area.

PATHOLOGY

The understanding of the local pathologic process has been handicapped by the paucity

FIG. 1 Medial aspect of right knee. (Left) Longitudinal incision to expose fascia. (Center) Incision through fascia lata showing thin gliding membrane containing veins that course over medial condyle of femur. (Right) Attachments of adductor magnus tendon and tibial collateral ligament. (Nachlas, I. W. Olpp J. L. Para-articular calcification (Pellegrini-Stieda) in affections of the knee, Surg., Gynec. & Obst. 81:208)

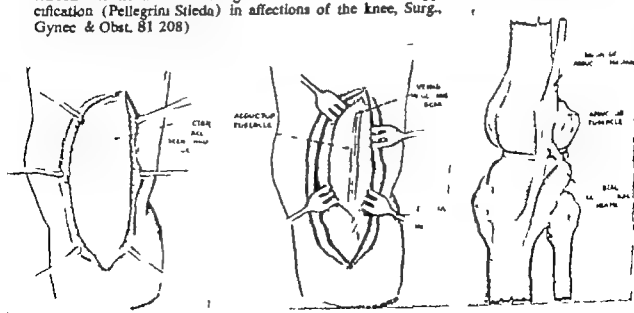




FIG. 2. Ossified mass overlying adductor tubercle. Even though photograph is reduced, organization of mass, with cortical periphery and trabeculated center can be recognized. (Nachlas, L. W., Olpp J. L. Para-articular calcification (Pellegrini-Steda) in affections of the knee, Surg., Gynec. & Obst. 81.209)

FIG. 3 Showing typical evolutive lesions (Kulow ski, J Post-traumatic para-articular ossification of the knee joint, Am. J Roentgenol. 47 399)



of operative explorations. Most of the information obtained has been derived from studies of the x ray films, but these are of only limited value so that there is still no unanimity of opinion as to the particular anatomic structure that is involved. In view of this, a review of the local anatomy may be helpful.

As one dissects the medial aspect of the knee one proceeds through the skin and the subcutaneous tissue to expose the firm fascia lata. Beneath this there appears a white glistening membrane that comes down the inner aspect of the thigh over the adductor magnus muscle to invest snugly the condylar expansion of the femur. This membrane is not firmly fixed to the bone but glides over it. In the fresh specimen this layer is sufficiently transparent to permit one to see several small blood vessels running longitudinally from above the adductor tubercle to disappear in the deeper structures at the level of the articular surfaces. Beneath this membrane one finds the tibial collateral ligament, the periosteum and the capsule

the adductor tubercle and the tendon of the adductor magnus (Fig. 1).

The bony surface that forms the base of the dissected area, i.e. the inner surface of the medial condyle is slightly convex in contour. A bony hump near the middle of this surface serves as an epicondyle which occasionally is found to be grooved horizontally so as to present two prominences. The lower of these is the point of attachment for the tibial collateral ligament, while the upper together with the proximal and posterior portion of the bony surface serves as the attachment for the adductor magnus muscles and the medial head of the gastrocnemius. The latter usually overlies a small bursa and at times contains a small sesamoid bone. A larger bursa separates this muscle from the internal hamstrings that run over this area.

It has been definitely established that the mass visualized by x rays is calcific in character. Calcification does not appear immediately after the injury. Benassi is said to have found a case in which there was a lapse of only 2 days, but while the interval

quite variable most people find that the mass appears between 2 and 4 weeks following the injury. It is quite likely that the calcifications found in periods shorter than this were produced by earlier trauma.

The mass usually begins with soft tissue swelling in which a slight amount of calcific deposit appears. This calcification may increase in size and eventually can recede. Complete absorptions of the mass have been reported.

Characteristically, the x ray shadow is seen on the anteroposterior view and is found to be a thin semilunar line overlying the adductor tubercle (Fig. 2). Usually it is about 2 cm. long and about 2 mm. thick. There is always a radiolucent area from 1 to 2 mm. in thickness between the bony shadow and the femur. Serial roentgenograms may show the mass enlarging, and, as a rule, the elongation takes place proximally while the thickness increases medially. As the line elongates, the curve of the semilunar shadow reverses itself so that an S-shaped configuration is developed (Fig. 3). The density of the shadow is usually that of a granular calcareous deposit which is thicker in the center and somewhat fuzzy on the edges. In more firmly established masses one finds definite increase in the density of the entire mass with rather sharply demarcated outlines.

The lateral view usually fails to reveal the mass. This is due to the fact that its shadow is masked by the much greater density of the entire condylar portion of the femur. Because of this the roentgenogram does not reveal the full extent of the lesion in its third dimension. However, one can make some deductions from this negative finding. One can assume that the mass does not extend behind the level of the condylar projection. This helps us rule out, as potential housing for the mass, those tissues which lie posterior to the bone. Therefore on the basis of the x ray findings one can localize the mass as follows: it lies external to the periosteum of the bone, proximal to the tibial collateral ligament and is embedded

in the soft tissues that cover the inner condyle. In view of the shape of the mass and its change from a semilunar to an S-shaped figure, one must consider the likelihood that it is molded by the gliding membrane described above. In other words, on the basis of the x ray findings one has reason to believe that the typical shadow is produced by a calcific mass lying immediately under the gliding membrane but separated from the bone proper by the periosteum.

This localization of the bony mass finds corroboration in some of the operative observations. Kulowski who operated on one patient found "This formation infiltrated the tendons of the adductor magnus and vastus medialis but was nevertheless clearly defined. It was intimately related to the internal lateral ligament but did not actually involve it." ¹⁰ Finder who divided his patients into 3 groups explored one knee in the group that had not only the characteristic Pellegrini Steda picture but also peripheral bony shadows. In this patient he found a bony mass in the central area of the ligament. Nachlas, in a typical Pellegrini-Steda knee, found a buttonlike discrete mass lying in the interval between the membrane and the condyle overlying the adductor tubercle. Its external surface was firmly attached to the overlying membrane, but its inner surface lay loosely on a thin fibrous bed that was granular in appearance. The mass did not invade the internal lateral ligament, the tendon of the adductor magnus, or the tissue of the adjacent muscles. A palpating finger directed toward it from inside the joint fell far short of reaching it. In other words, it was not housed within any of the organized anatomic structures but had made a place for itself in the latent space between the gliding membrane and the periosteum.

Histopathologically it is generally believed that the radiopaque material is at first deposited in a granular calcific form. However, the specimens that were obtained in the 3 operative cases just mentioned, show well formed bone tissue. Kulowski states that the central portion is usually osseous.

but sometimes calcific. His specimen is described as follows: "The tissue grossly presented an osseous body embedded in dense ligamentous tissue. The tendinous tissue near the bone is edematous (preosseous edema?) and the cells are round rather than spindle shaped but still parallel in arrangement. This layer of edematous tissue merges with the bone. Fibers of ligament merge imperceptibly with the fibrils of the bone. The bone is living cellular bone almost entirely composed of fibrous bone. The architecture closely resembles that seen in the very thin compacta over the metaphyses of a long bone. Along the walls of the larger vessel spaces is a row of osteoblasts and bone apposition is occurring. Often there is a thin layer of lamellar bone (of osteoblastic origin) along such a vessel space. This is demarcated from the fibrous bone by a blue cement line. Concentric layers of lamellar bone are found around some of the smaller vessel spaces. In a few areas osteoclastic resorption is taking place. Most of the vessel spaces are filled with a loose cellular vascular connective tissue."¹⁰

With regard to the pathogenesis, a number of theories have been suggested. To Pellegrini is attributed the belief that the bony mass is made by periosteal proliferation and metaplasia of the medial collateral ligament. Stieda felt that it is due to an avulsion fracture of the epicondyle with subperiosteal proliferation of bone. Terrier believed that it is produced by the calcification of a blood clot between the adductor magnus and the vastus internus. Kulowski considered the lesion as a form of myositis ossificans.

Johannes Volkmann,² who also separates the shadows into three groups, of which one group seems to conform to the classical picture attributes the mass to a "metaplastic process either in the tendinous tissue or in an ossifying hematoma." Nachlas and Olpp offered the opinion that the small vessels on the medial aspect of the knee rupture to form a collection of blood that is trapped in the space between the gliding membrane and the underlying bone. The hematoma

undergoes calcification and subsequent conversion to bone.

It is noteworthy that as pointed out by Shanks and Kerley deposits resembling those of the Pellegrini-Stieda lesion may be found in the soft tissues on the posterior surface of the tibia or medial malleolus.¹¹

CLINICAL PICTURE

The patient usually comes in complaining of pain in the knee. A history of an injury can ordinarily be obtained. Frequently one learns that the patient has had some antecedent trouble with the knee and that the joint has "flared up" from time to time but has had periods of remission. The current episode may have begun a few weeks earlier but the discomforts in the knee did not subside. A limp may have developed. On examination one often finds evidence of an articular involvement. There is swelling, capsular thickening and fluid increase in the joint. Over the medial aspect of the joint one may note some thickening on palpation over the condyle. Pain can be elicited. A bony prominence may be present but it is usually difficult to determine by the examining finger. When the joint is flexed the patient may complain of pain over the epicondyle but this is encountered only occasionally.

The positive diagnostic criterion is obtained by x ray examination when it reveals the calcareous deposit. If in the first few weeks after the injury the film does not demonstrate the deposit, one must recall that there is a latent period in its development. If one suspects that a lesion is present, or if the joint fails to follow the normal pattern of recovery the x ray examination should be repeated at 2 week intervals. After the existence of the deposit has been established serial roentgenograms are indicated to help one follow the course of the mass.

Since the symptoms about the knee need not all be attributable to the calcified mass, one must differentiate between those produced by an articular involvement and this extra articular lesion. In this connection it must be remembered that the trauma that

led to one may also lead to the other. Intra-articular pathology will present such evidences of synovitis as ballottement, thickening of the capsule and local tenderness. In the joint one may be able to feel loose bodies or cysts of the cartilage. In the Pellegrini-Stieda lesion the swelling and the tenderness will be limited to the medial aspect and be maximal near the adductor tubercle. The point of greatest tenderness in the articular lesions is likely to be at the level of the articular surface, often over the medial meniscus. Differentiation from extra-articular pathology such as semimembranosus bursitis or direct contusion, may be more difficult. Careful attention to the exact location of the swelling and the tenderness will be helpful here. In evaluating the roentgenograms one must ascertain the presence of the radiolucent zone. Sometimes it will be found necessary to alter the projection slightly to bring this out. The radiopaque area may vary considerably in size, shape and density, but the line of separation should be present.

TREATMENT

When the patient presents himself with his injured knee, interest is usually centered on the articular damage. The treatment that is indicated for this is ordinarily some form of physiologic rest. This treatment is beneficial to the Pellegrini-Stieda lesion, too, and in the early precalcific stage is all that is required. The rest allows the swelling that serves to promote the deposit to subside and constitutes good prophylaxis. If, in spite of the protection, calcification should develop, roentgen therapy may be helpful. Limitation of activity is desirable. In fact, under this regimen the calcification may reach its peak and recede to a complete resorption without the use of any other therapy. When the calcific deposit has been changed to an osseous structure, radiation therapy is not likely to make it disappear.

When the Pellegrini-Stieda lesion is found in the established or chronic stage, the need

for active treatment must be determined on the basis of the symptoms. In most instances the deposit may be allowed to exist without any therapeutic measures. It remains *in situ* without producing symptoms. In other instances, particularly when it is so large that it acts as a mechanical interference to the gliding of the medial structures as the knee flexes and extends, it may be necessary to remove it. Under such circumstances, care should be taken to allow the mass to become stabilized, i.e. to reach maturity. Attempts to excise the mass prematurely have resulted in recurrences of calcific deposits which have exceeded the original mass in size. The operation should be performed with minimal trauma to the adjacent tissues and care should be observed to leave a dry field. Postoperative immobilization for 3 weeks is desirable. When operative intervention is used on properly matured lesions, one may expect good results.

It must be re-emphasized that in most patients in whom the Pellegrini-Stieda shadow is found as an incidental observation, no special treatment is needed.

SUMMARY

The soft tissues on the medial aspect of the knee are so arranged that under proper conditions they predispose to the formation of a local calcified mass. The precipitating factor is trauma, which produces a hematoma that undergoes calcification. This mass may disappear spontaneously or may change to bone. The presence of the deposit rarely calls for surgical intervention, but occasionally it is so large that it impedes the normal movements of the joint. Under such circumstances, after the deposit has become stabilized, it may be excised with the anticipation that a cure will be obtained.

REFERENCES

1. Pellegrini, A. *Clin. mod.* 2:433 1905.
2. Stieda, A. *Arch. klin. Chr.* 85:815 1908.
3. Volkmann, J. *Monatsschr. Unfallh.* 52:353 1949.

- 4 Kulowski, J. Am. J Roentgenol. 47 392, 1942.
- 5 Nachlas, I. W and Olpp J L. Surg Gynec. & Obst. 81 206 1945
- 6 Pellegrini, A. Arch ital. chir 53 501 1938
- 7 Finder J G J.A.M.A 102.1373 1934
- 8 Russell, D A., and Smith, C. D Radiology 46 351 1946
- 9 Wetzler S. H., and Elconin D V Am. J Surg 27 245 1935
- 10 Kulowski, J J.A.M.A. 100-1014 1933
- 11 Shanks, S C., and Kerley P A Textbook of X ray Diagnosis ed 2, vol 4 Philadelphia, Saunders, 1950

SECTION II

GENERAL ORTHOPAEDICS

Giant-Cell Tumor of Bone and Its Differential Diagnosis

WILLIAM C. HERRICK, M.D.,* AND
HENRY L. KAZAL, M.D.†

INTRODUCTION

The difficulties in diagnosing and treating giant-cell tumors of bone have a familiar ring to all those interested in bone disease particularly to those interested in bone tumors. The literature contains much discussion on the subject as well as a number of reliable series of cases. One need only follow the history of giant-cell tumor from 1837 when it was described by Warren, until its present-day grading, as described by Jaffe and Lichtenstein, to appreciate the controversies that have arisen. One of these is whether the lesion is benign or malignant, while another, even more basic, is whether or not it is a tumor at all. The microscopic examination of lesions similar clinically and roentgenologically containing multinucleated large giant cells has led to their misdiagnosis as giant-cell tumors of bone.

It is the purpose of the authors in this chapter to sketch the natural history of giant-cell tumor from observations gleaned from 30 cases drawn from the files of Jefferson Medical College Hospital as well as from the abundant medical literature on the subject. Included in the chapter are the clinical

manifestations, the roentgenographic findings, the gross appearance, the microscopic details and the approach to the treatment.

One of the greatest problems in the differential diagnosis of giant-cell tumor of bone lies in segregating it from some of the more closely allied lesions of bone. In reviewing our own cases that were diagnosed as giant-cell tumors, for example, we were able to find several ossifying fibromas, an aneurysmal bone cyst, a reactive bony lesion following trauma, a chondroblastoma and a chondromyxoid fibroma. This is in no way a criticism of our predecessors 25 to 30 years ago who attempted to make the diagnosis when the issues were clouded by confusion and at a time when insufficient data had not yet been accumulated. However, this is no longer true as a result of the efforts of such men as Bloodgood, W. B. Coley, Ewing, Stewart, Stout, Jaffe and Lichtenstein, and Codman. By consolidating the present-day concepts in diagnosis and management, we hope that some of the still-existing problems can be solved.

HISTORY

From a perusal of the literature it is difficult to be sure that the early descriptions were concerned with what is known today as giant-cell tumor or whether they dealt with its variants. Certainly Paré's account

* Assistant Professor of Pathology, Jefferson Medical College and Assistant Director of Clinical Laboratories, Jefferson Medical College Hospital.

† Resident in Pathology, Jefferson Medical College and Hospital.

of giant-cell tumors occurring in the maxilla might well have been ossifying fibromas or osteitis fibrosa cystica of hyperparathyroidism, because giant-cell tumors are uncommon in the maxilla. Warren (1837) is credited more rightfully with the first description of the giant-cell tumor for although he described lesions in the maxilla, he also described them as occurring in the epiphyseal ends of the long bones.

Regardless of where the credit for the first description lies, the development of the present-day concept of the giant-cell tumor certainly has been a "stormy" one with bitter opponents each believing his own concept to be true. Widespread amputation was practiced for many years and was augmented greatly by the use of the term "sarcoma" as a suffix after myeloid or giant cell. Staunch defenders of an inflammatory origin and nature were more numerous in past years, although a few still hold to the doctrine.

Although Paget, as early as 1853, no doubt had meticulously described the true giant-cell tumor and furthermore was of the opinion that even though many giant-cell tumors were benign a few went on to malignancy, his colleagues and followers were not entirely willing to accept his concept. For example, a few years later we read that Virchow contended that most giant-cell tumors were malignant. Nélaton (1863) incorporated much of our present-day knowledge of the gross microscopic and clinical findings found in our present-day literature but was very emphatic in stating that all tumors with giant cells must be regarded as benign in the younger age groups even though there was an odd mixture of fibroblasts and cartilage cells. However, with advancing age of the patient and alterations in the histologic structure, he was not sure that the lesion would remain benign. Furthermore, he felt strongly that simple curetting of the tumor might be inadequate and that resection or amputation might have to be resorted to in the final analysis.

However, Gross countered, in 1879, with an accurate analysis of some 70 cases of

giant-cell tumor with descriptions of the gross and the microscopic findings. He insisted that the tumors were benign but admitted difficulty in separating some of the lesions from vascular osteogenic sarcomas.

One of the great milestones in the present-day concept in discovering the true nature of the lesion but also in arriving at the proper mode of treatment was afforded by Bloodgood, in 1919, when he reported 47 cases treated by curettage without recurrence. He employed and encouraged the use of the term "giant-cell tumor" rather than giant-cell sarcoma in a day when widespread amputation was being practiced in the treatment of such cases. Following this, together with the efforts of W. B. Coley, thorough curettement was practiced more often, although overzealous amputation still was encountered occasionally.

Ewing cited a few instances of spontaneous cure but also stated that in most cases in which the giant-cell tumor was left to run its course there was a "natural termination in death from hemorrhage and infection." Ewing not only described accurately the gross and the microscopic findings of giant-cell tumor but also recognized aneurysmal giant-cell tumors, benign calcifying giant-cell tumors (Codman's tumor, chondroblastoma), and malignant giant-cell tumors.

British writers, as well as others, have continued to use the term "osteoclastoma" primarily because of the writings of M. J. Stewart and R. A. Willis, who believe that the tumor arises from osteoclasts. Stewart first suggested the name "osteoclastoma" in 1922, and it has been adopted by most British pathologists. Certainly there is much to be said in favor of their comments on the subject. The multinucleated giant cell seen in this lesion certainly has an etiology and it does resemble somewhat, an osteoclast.

Yet to be separated in some detail were the so-called giant-cell tumor variants, such as the brown tumors of hyperparathyroidism, the aneurysmal bone cysts, nonossifying and ossifying fibromas of bone, chondroblastomas, and even chondromyoid fibromas.

configuration and, if left uninterrupted, will progress on to death by hemorrhage and/or infection (Ewing). Complete healing and cyst formation following pathologic fracture has been described (Jungling, Martland). In malignant-cell tumors pulmonary metastasis does occur but it develops later in the course of the disease. Pathologic fractures occur in 14 per cent of the cases (Geschickter and Copeland). Physical examination reveals swelling and disturbed joint function as the outstanding findings. There is tenderness to deep palpation but actual pulsation of the tumor is uncommon even though the periosteum covering the hemorrhagic tumor is very thin. The intact articular cartilage greatly aids in preventing joint effusions.

AGE

It is fairly well agreed that approximately 90 per cent of giant-cell tumors occur in the third, the fourth and the fifth decades of life (Geschickter and Copeland). Although the average age is between 30 and 33 years, the greatest incidence lies between 20 and 50 years. Jaffe has seen one case in which the patient was 18 years old, and one in which he was 19. Certainly giant-cell tumors are rare below the age of 20 years but may be seen any time after the closure of the epiphysis.

SEX

According to Christensen's series males are affected about equally with females—males 170, females 192 (47 to 53%).

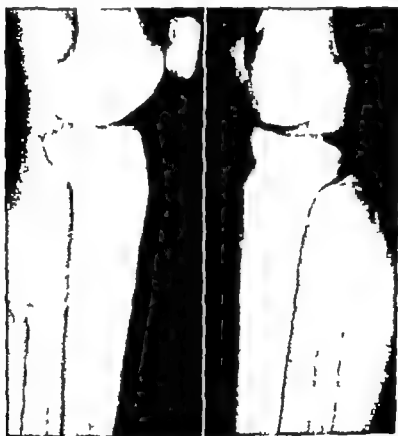


FIG. 1 (Left) Roentgenogram of a giant-cell tumor situated in the upper end of the fibula of a 28-year-old male showing an area of rarefaction with thinning, expansion and partial destruction of the overlying cortex.

FIG. 2 (Right) Roentgenogram of a similar giant-cell tumor in the upper end of the fibula of a 19-year-old female. The almost complete absence of periosteal new bone formation is notable. The multiloculated appearance is seen frequently.

LOCATION

According to Jaffe and Lichtenstein, approximately 60 per cent of giant-cell tumors occur in the lower end of the femur, the upper end of the tibia and the lower end of the radius. Christensen's figures are much the same. Sites other than those mentioned above (in decreasing order of frequency) are the proximal end of the humerus, the distal end of the ulna and the proximal end of the fibula. Rarely does the growth occur in the small bones of the feet, the calvarium, or the vertebrae, although Willis states that 25 per cent of the giant-cell tumors occur in the head and the trunk (especially the pelvis and the vertebrae). We feel that this percentage is unduly high. Most of our cases occurring in the head eventually were reclassified as ossifying fibromas and reactive bone changes. We have one case of a very large giant-cell tumor occurring in the ilium.

Acherman cites 3 cases with lesions in the bones of the hands which resembled greatly the giant-cell tumor but in all of which were eventually found adenomas of the parathyroid glands. Not only may lesions in this locale be the brown tumors of hyperparathyroidism, but also enchondromas are common here.

Cystic lytic lesions occurring in the vertebrae may grossly be hemorrhagic and resemble giant-cell tumor histologically but are more likely to be the aneurysmal bone cyst or hemangiomas or even myelomas.

A giant-cell tumor occurring with Paget's disease has been reported by Jaffe.

When cases of multiple giant-cell tumors are reported, then careful biochemical studies should be done to rule out hyperparathyroidism.

Giant-cell tumors occur so consistently at the ends of long bones in the region of the epiphysis that the name "epiphyseal giant cell tumor" has been recommended. Coley describes unusual giant-cell tumors occurring in the shafts of long bones. We have not encountered such a lesion nor have we en-

countered one instance of multiple giant-cell tumors.

ROENTGENOGRAPHIC FINDINGS

The successful interpretation of this tumor is better than for many varieties of bone neoplasms. It is true that the characteristic "soap bubble" effect is commonly present, but there are exceptions (Figs 1 and 2). The characteristic roentgenogram of a giant-cell tumor will show a radiolucent mass involving the epiphyseal region of a long tubular bone. Trabeculae course through its center giving it a "bubble" effect. The lesion expands the walls of the ends of the bone eroding the cortex and the periosteum so that only a thin shell remains visible on the roentgenogram. Seldom does it spread into the shaft, although it may involve the metaphysis. The neoplasm may extend to



FIG. 3 Roentgenogram of a giant cell tumor located in the upper end of the humerus of a 23 year-old female. The epiphysis and the metaphysis have been destroyed. The cortex has been expanded and thinned, and there is little periosteal new-bone formation. The characteristic "soap bubble" appearance is well shown. The location of the lesion is unusual.



FIG 4 Giant-cell tumor of the tibia. The expanded end of the bone shows extensive destruction with trabeculation and thinning of the overlying cortex.

fill the condyles and approach the articular cartilage but does not destroy the latter. Pathologic fracture may be present in 14 per cent of the cases (Geschickter and Cope-land). Rarely will there be displacement.

Other more serious lesions occurring at the ends of long bones and resembling the giant-cell tumor are chondrosarcoma and fibrosarcoma. Therefore, no matter how "typical" the x ray film may be of giant-cell tumor the diagnosis should not be final until tissue diagnosis has been made. Certainly no mode of therapy should be instituted until the diagnosis is confirmed histologically (Fig 3).

GROSS PATHOLOGY

The gross pathology of this lesion although fairly characteristic varies with the size of the tumor, the age, the amount of hemorrhage, necrosis and whether or not

pathologic fracture has taken place. Usually one sees a markedly enlarged end of a long bone, the periosteum distorted and widened by a bulging mass in the center of the bone. On cut surface in the full-blown "typical" state, an expanding, bloody, hemorrhagic mass, with necrotic solid and cystic areas occupies the end of a long bone. The lesion looks like a large loculated hematoma within the confines of the thin bony periosteum, having destroyed the epiphyseal area. Occasionally however the whole mass may not be bloody or have the typical maroon color but may have white patches representing more fibrous areas. The cut surface, as one would expect, is very friable. There is no capsule around the giant-cell tumor but its expanding nature compresses the tissue outside of it so that it may appear to have one (Fig. 4).

Early in the neoplasm one might find a few remaining trabeculae coursing through the tumor. As the giant-cell tumor grows, destroying and resorbing bone spicules, the trabeculae disappear and the periosteum becomes a thin membrane. The articular cartilaginous end plate is rarely destroyed, although the subchondral end plate may be eroded. A few cases where the articular cartilage was destroyed have been described but this is uncommon. A lesion which is left to make its course without surgical intervention, may not only develop a pathologic fracture but may also bulge into the adjacent soft tissues.

MICROSCOPIC FINDINGS

In a review of 30 cases of giant-cell tumor diagnosed in our own hospital collected over the past 25 to 30 years, the need is apparent for careful microscopic examination of the giant-cell tumor of bone. Because of the unnecessary accent placed on giant cells which many bone lesions have: nonosteogenic fibroma, chondromyxoid fibroma, unicameral bone cyst, aneurysmal bone cyst, osteitis fibrosa cystica of hyperparathyroidism and chondroblastoma have been mistaken for the problem child of bone tumors—the giant

Sprains

GARRETT PIPKIN M.D *

Scientifically the subject of "sprains" is difficult to discuss because such a diagnosis is no longer recognized. If one looks up "sprains" in the *Index Medicus* one finds "see individual joint such as wrist, ankle" subtitles read "wounds and injuries of." In other words there is no such thing pathologically as a sprain and the scientists are insisting that the diagnosis be made more nearly accurate and they want the anatomic structure involved named specifically e.g., a rupture of the deltoid ligament.

The average doctor and the laity are not concerned with scientific definitions and will continue to use the terms "strain" and "sprain" in spite of what the *Index Medicus* lists.

According to Blue Shield statistics for the Kansas City Missouri area covering their fiscal year ending Dec 31 1952 the number of months of exposure to risk for all their members was 3 653 411. During this year they allowed payment for 114 698 claims. Included in this total were 1 486 sprains representing approximately a 1 per cent ratio. Their next largest allowance in their "Bone and Joint Classification" was for Fracture of the Clavicle which amounted to 257. "Colles Fracture" was third totaling 210 payments. The average of payments made to participating physicians covering sprains was \$5.33 to which must be added the overhead

for clerical processing. All sprains during the fiscal year of 1952 cost Blue Shield \$7,095.52. The cost to Blue Shield for 257 fractured clavicles was \$11,358.²

Insurance experts have pointed out that the individual would be financially ahead by assuming liability for sprains himself. They substantiate this statement with the following reasons: (1) that his recovery from Blue Shield is so small and (2) that by eliminating the cost of processing these small claims, Blue Shield would be in a position to lower premiums.

Several of the major insurance companies in this area issue policies which include payment for sprains. They also state that they have trouble processing these claims for sprains since there is such a wide variance in the reports made by the doctors covering these injuries. One large company here reports that they have processed a claim in excess of \$1,500 for which they received no diagnosis other than "sprain."

The purpose of the foregoing list of statistics has been to show that strains and sprains are a real problem to the insured (the injured person) to the doctor and to the insurance companies. Any attempt made to resolve these difficulties must include some analysis of the injuries that are usually diagnosed as "strains or sprains."

As late as 1930 acceptable medical terminology defined a sprain as "a wrench or a strain resulting in stretching or laceration of the soft parts without external wound." The outcome of a "sprain" therefore depends

Attending Orthopedic Surgeon, Kansas City General Hospital and Kansas City St. Joseph Hospital. Associate Editor *Missouri Medicine and Clinical Medicine*.

cell tumor. The salient features of the histologic picture in the giant-cell tumor are (1) stroma cell and (2) giant cell.

The giant cells are large and vary from 50 to 100 microns in diameter. They show numerous nuclei that number as high as 100 in some cases but usually disclose from 30 to 60. The nuclei are prominent with distinct nuclear membrane and a reticular network. The cytoplasm is eosinophilic. The number of giant cells per high-power field may vary a great deal from field to field. When giant cells are small and contain few nuclei and are sparsely scattered through the tumor, one should hesitate in making the diagnosis of giant-cell tumor.

The stromal cell is the more essential cell for the diagnosis of this tumor and is the key to the prognosis through the histologic grading as described by Jaffe and Lichtenstein. The stromal cell has one nucleus, as a rule, and looks much like young connective tissue cells. The contour of the cell is spindle-shaped with a central prominent nucleus and a moderate amount of chromatin and a centrally placed nucleolus. The cytoplasm stains lightly eosinophilic. It can be noted also that the nucleus of the stromal cell in a given tumor resembles highly the nuclei contained within the giant cells. According to the compactness, the degree of cellularity and the amount of atypism present afford the basis of histologic grading of these tumors. Furthermore, in the grading of these tumors, the most ominous area should be used for the corresponding grade regardless of the fact that much of the tumor may appear to be benign.

GRADE I

Giant-cell tumors in Group I show the stromal cells to be loosely spread through the background and show almost no atypism. Stromal cells will be spindle-shaped or round and oval. There will be little or no variation in size although shape may vary and for the most part staining qualities will be about the same. Occasional mitotic figures are found. Rarely is osteoid tissue produced by

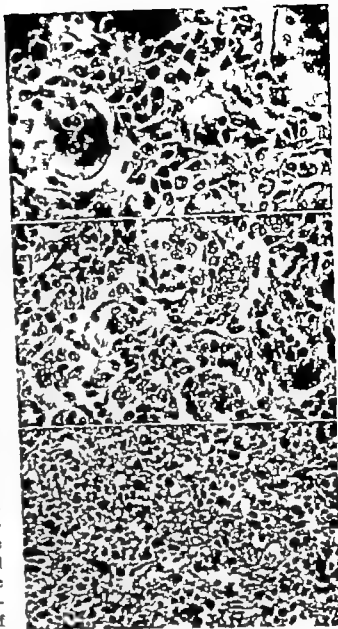


FIG 5 (Top) Photomicrograph of a giant-cell tumor of Grade I. The loosely arranged stromal cells are uniform in size with minimal atypism. Giant cells with numerous nuclei are plentiful (X 400).

FIG 6 (Center) A giant-cell tumor of Grade II, showing a more cellular and compact stroma with some atypism of the cells. The giant cells, which are still numerous in some fields, show nuclear atypism tending to parallel that seen in the stromal cells. (X 400).

FIG. 7 (Bottom) Grade III giant-cell tumor demonstrating a sarcomatous type of stroma. In addition to the occurrence of large irregular anaplastic nuclei within the stromal cells, there is a markedly irregular arrangement of these cells. The giant cells are inconspicuous with a paucity of nuclei which are typical. (X 260).

the stromal cell. The connective tissue framework is vascular and areas of hemorrhage are common.

GRADE II

These tumors show definite atypical cells, but to a minimal degree. Whereas the Grade I giant-cell tumors have a loosely packed stroma, Grade II giant-cell tumors show a greater degree of cellularity and are more closely compacted. Mitotic figures are more prominent and increased in number. The stroma is beginning to take on more of a sarcomatous appearance.

GRADE III

Grade III giant-cell tumors possess a definitely sarcomatous stroma. Atypism is marked, as is hyperchromatism. Many of the stromal cells will contain two and three nuclei. The stromal cells are more numerous than in Grades I and II and more compact. As the degree of malignancy of the giant-cell tumor increases, the size and the number of nuclei in the giant cells decrease. The nuclei in the giant cells are as atypical as the nuclei in the neighboring stromal cells.

According to the experience of authors such as Jaffe and Lichtenstein, given a sizeable series of giant-cell tumors, one half are likely to have a favorable outcome whether treated by x rays or surgery. Approximately one third are more apt to be aggressive, recurring after treatment and may eventuate in amputation. Fifteen per cent are frankly malignant and are likely to metastasize. Rarely is a giant-cell tumor found to be Grade III on initial examination. Grade III giant-cell tumors are more apt to occur after repeated and unsuccessful treatment of Grades I and II giant-cell tumors (Figs 5, 6, 7).

DIFFERENTIAL DIAGNOSIS

Many lesions have only recently been separated away from the true giant-cell tumor. Some of these lesions are listed below with differential points in diagnosis.

ANEURYSMAL BONE CYST

Jaffe has described aneurysmal bone cyst as "an essentially solitary, localized, and expanded fibrous lesion, honey-combed by an enormously dilated, plexy form vascular bed."

Clinical. Aneurysmal bone cyst occurs more commonly in young adults and children. It is seen more commonly in females than in males at a ratio of 2 to 1.

The lesions are usually located in the vertebrae or the femur. When in the long bones, it involves one end of the shaft but spares the epiphysis. Occasionally it is seen involving the tarsals, the metacarpals, the phalanges and flat bones as well as the calvarium. In two cases described, in the recent Mayo Clinic Series, the second cervical vertebra was involved.

Pain, swelling, tenderness and limitation of motion are the outstanding symptoms. They certainly are not diagnostic but merely indicate pathology of some nature in this area. If the lesion involves the vertebrae, it may encroach on the spinal cord so as to produce neurologic signs and symptoms.

Roentgenograms. X-ray films can show a rather typical appearance of this lesion and this is the case more commonly. A cyst-like area is seen eccentrically placed, expanding the cortex and partially absorbing it. The lesion is defined by a sharply etched rim of periosteal bone. The center of the mass is radiolucent, with fine trabeculations not unlike the "soap bubble" effect commonly attached to the giant-cell tumor. The cyst has a "blow out" appearance in many cases.

Pathology. Upon unroofing the thin layer of periosteal bone covering the cyst, blood will well up from the center of the mass. This blood is old and appears to be stagnant; it does not spurt or pulsate. The blood spaces are separated by their fibrous septal walls.

Microscopic examination reveals a field somewhat like that seen in the giant-cell tumor. The septal walls do not appear to be those of blood vessels. Occasionally osteoid

and nonlamellar, coarse fiber bones are seen in the septal walls. The stromal cells are spindle shaped with prominent nuclei and there is a scattering of multinucleated giant cells. More solid nonbloody areas may have an abundance of fibrous tissue in whirls and spicules of osteoid, looking much like fibrous dysplasia. There are two types of intra osseous fibromas of bone both have been confused with giant-cell tumor of bone; one shows ossification while the other does not.

NON-OSSIFYING FIBROMA OF BONE

Synonyms are giant-cell tumor variant of bone cyst, osteitis fibrosa healing variant of giant-cell tumor, occasionally xanthoma or xanthogranuloma of bone.

Clinical. Non-ossifying fibromas of bone occur in a younger age group than the giant cell tumor, namely 8 to 16 years and the sexes are equally divided (Jaffe and Lichtenstem). The lesion is seen at the ends of long tubular bones and prefers the lower extremity to the upper. It is seen in the region of the metaphysis. Cutaneous pigmentation and endocrine changes are lacking.

Roentgenograms will reveal a lytic lesion in the ends of the long bones, eccentrically placed and occasionally occupying the entire end of the long bone. Neither the periosteum nor the epiphysis is invaded. If the tumor grows to any great size bulging of the shaft will be noted.

Pathology. The bone shows a general fusiform enlargement in the area. On cut surface the nonossifying fibroma tends to be loculated and show yellow moderately dense tissue.

Microscopic examination reveals a rather clear-cut picture of dense whirls of fibrous tissue in tight curvilinear whirls. Multinucleated giant cells are seen in many fields, but for the most part they do not have as many nuclei as those in the giant-cell tumor. The giant cells are limited in the tumor to areas of hemorrhage signifying phagocytic function. These giant cells, together with histiocytes and foam cells, give rise to such terms

as solitary xanthomas or xanthic variant of giant-cell tumor.

OSSIFYING FIBROMAS

Ossifying fibromas were first described by Phemister and Grimson. This type of intra osseous fibroma is claimed by such authors as Schlumberger to be identical with fibrous dysplasia.

Clinical. For the most part, ossifying fibromas are limited to the jaw. Review of our cases of giant-cell tumors in the head and the neck revealed several ossifying fibromas. Sherman and Sternbergh's series of 11 cases showed 6 in the mandible and 5 in the maxilla. All were monostatic. The patients complain of facial asymmetry also difficulty with occlusion of the teeth due to bulging mass.

Roentgenograms reveal an essentially destructive process in the medullary portion of the bone. The lesion expands in all directions and periosteal reaction is conspicuously absent.

Pathology. Gross examination shows a lobulated fibroma of bone, usually small, but it can grow large enough to cause deformity. Microscopically the tumor shows—aside from dense fibrous tissue—multiple spicules of bone scattered through the tumor at various levels. Again around areas of hemorrhage giant cells will be seen but the phagocytes and the spicules of bone together with the dense fibrous tissues readily differentiate this from giant-cell tumor of bone.

CHONDROMYXOID FIBROMA OF BONE

This is another lesion that in recent years has been separated away from the giant-cell tumor as a distinct entity in its own right (Jaffe and Lichtenstein 1948). The lesion has been misdiagnosed more commonly as chondrosarcoma than giant-cell tumor of bone. By definition, it is a benign tumor derived from cartilage forming connective tissue, with cartilaginous and myxomatous tissues predominating and possessing occasional foci of multinucleated giant cells.

Clinical Although many of the cases described thus far are at the ends of long tubular bones already it is becoming apparent that they may occur in the small bones of the feet, the ribs and the pubic bones.

Symptoms are not remarkable but rather similar to any tumor growing and expanding a bone, namely, pain tumor and disturbance of function of the neighboring joint.

Roentgenograms reveal a partially lytic expanding mass showing rarefaction and pseudotrabeulation. In the long bones the tumor involves the metaphysis, is eccentric and destroys the overlying cortex. In the metatarsal bones the tumor may grow to fill the entire bone.

Pathology The tumor may grow to considerable size—8 x 5 cm (Lichtenstein). The cut surface of the tumor has a rubbery white texture and a cartilaginous appearance quite unlike the bloody appearing giant-cell tumor.

Microscopic sections reveal myxoid and cartilaginous tissue with occasional foci of multinucleated giant cells. However the giant cells are scarce and tend to be smaller with fewer nuclei than those seen in the giant-cell tumor. In addition the giant cells are limited to areas around blood vessels where extravasation of blood has occurred. The background cell is spindle- or multipolar-shaped in a myxoid intracellular matrix.

BENIGN CHONDROBLASTOMA OF BONE

Benign chondroblastoma of bone (Jaffe and Lichtenstein 1942) has the following synonyms: benign calcifying giant-cell tumor (Ewing 1928) and epiphyseal chondromatous giant-cell tumor group with certainty until as late as 1931 when Codman reported in detail 11 cases occurring in the head of the humerus.

Clinical. For the most part, chondroblastomas occur below the age of 20 years and from 80 to 85 per cent will occur between 13 to 17 (Jaffe). Six of Codman's 9 cases and all of Jaffe and Lichtenstein's cases were males. All of Codman's cases were in the

head of the humerus whereas Jaffe had 4 in the lower femur, 3 in the upper tibia, 1 in the lower tibia, and 1 in the upper humerus. Chondroblastomas, like giant-cell tumors, originate in the epiphysis and may extend into the adjacent metaphysis.

Following an insidious onset, symptoms common to tumors arising in this locale follow in 9 months to a year: namely, pain, swelling and disability referable to the nearest joint.

Roentgenograms reveal an oval eccentric lytic area in the epiphyseal or juxta-epiphyseal area. There is a mottled appearance instead of the "soap bubble" trabecular appearance. The mass looks like ground glass. Although the tumor is radiolucent on roentgenograms it is not as radiolucent as the giant-cell tumor. The round lytic tumor mass commonly will cross the epiphyseal plate. New bone may be laid down by the periosteum.

Pathology Chondroblastomas are smaller tumors than giant-cell tumors. In general, they measure from 1 to 6 cm. in diameter (Jaffe). Usually the epiphyseal plate is destroyed, and the articular cartilage may be damaged but this will be only minimal. More well-preserved portions of the tumor will be grayish brown and firm. Areas of calcification are present, as well as cystic areas of hemorrhage and softening.

Microscopic examination reveals multinucleated giant cells around areas of hemorrhage. It is these giant cells that apparently have made the diagnosis confused with giant-cell tumor of bone. However giant cells in chondroblastoma are inconstant and may even be absent in areas. The background stromal cells offer more definite differences for here we find fields of polyhedral cells. The cells do not vary greatly in size or shape. The cells are compact with a rather large nucleus. The cytoplasm is lightly staining and eosinophilic. The highlight of findings is the focal area where calcium is sprinkled between the cells. At first, light deposits and then more heavily calcified areas are found. As the calcifica-

tion becomes heavier the stromal cells in this area become necrotic and die. This, in turn, is replaced by light hyaline connective tissue foci or focal areas of cartilage. It is best in preparing sections of this tumor not to decalcify it lest the histologic details be obliterated.

UNICAMERAL BONE CYST

Clinical. Unicameral bone cyst tends to involve the long bones of children or young adults. Geschickter and Copeland's series of 205 cases revealed that most patients were between 10 and 15 years of age. However, the solitary cyst can occur in older individuals as well as younger.

The average time for development of the symptoms is 2 years (approximately 6 months longer than that for giant-cell tumor). Pathologic fracture is the outstanding introductory clinical finding in 45 per cent of cases (Geschickter and Copeland). Following pathologic fracture the cyst may heal and 50 per cent of those that develop fracture have no subsequent symptoms. In the remaining cases, a new cyst or cysts may develop. Pain and swelling are conspicuously absent as is disability.

Roentgenograms. Almost all solitary cysts begin in the metaphysis but do not cross the epiphyseal plate. This cyst is usually seen on the diaphyseal side of the epiphyseal plate in young individuals before it closes in comparison with the giant-cell tumor which is seen on the opposite side of the epiphyseal plate after it closes. Examination of the roentgenograms reveals a solitary radiolucent mass fairly sharply outlined, showing thinning of the cortex and pseudotrabeculation.

Pathology. Unicameral bone cysts are characterized by a solitary cyst lined by a hyalinized, thick, connective tissue membrane. This membrane is avascular and varies in thickness. The cyst may contain a few to several cu mm of brown serous fluid. Following pathologic fracture this fluid will be bloody and increased in amount. Occasionally, instead of containing fluid the

cyst will be filled with a meshwork of light fibrous tissue.

Microscopically a thick hyaline lining of the cyst is noted. Giant cells are present but less numerous than seen in giant-cell tumor. The giant cells are more localized around blood vessels and areas of hemorrhage. The stroma is dense and fibrous.

Treatment. When giant-cell tumors occur in sites accessible to surgery, then the tumor may be completely resected. Where surgery involves marked loss of function of a limb although amputation is not done irradiation should be considered. Neither type of therapy should be attempted prior to biopsy and a histopathologic diagnosis.

THE BIOPSY

The biopsy may be open surgical or the needle aspiration biopsy. The open surgical biopsy does not preclude later surgical treatment if radiation is preferred. At the time of the biopsy a frozen section can be performed with safety of differentiating a giant cell tumor from a sarcoma of some type. Grading the stroma, however, must await paraffin section. Disadvantages of open surgical biopsy do exist because time must elapse to permit wound healing if radiation is contemplated, and subsequent herniation through the incision is possible.

The needle biopsy has been used successfully in many hospitals as well as at our Institution in aspirating bony lesions. It is quite possible to obtain tissue by passing a needle through the thin overlying periosteum and have sufficient material to diagnose the lesion correctly. Results can be given in 10 to 30 minutes on the smear or within 4 hours on the clot. Little time is wasted and an unnecessary operation is avoided as well as additional expense to the patient. If the diagnosis cannot be made by needle biopsy then an operative biopsy should be performed. If the diagnosis is made by the needle biopsy the choice of therapy can be made with a minimal loss of time.

Radiation therapy has a definite place in the treatment of the giant-cell tumor.

indications have been stated already. Irradiation is capable of controlling effectively giant-cell tumors of the less aggressive type. No reliable series of radiation therapy to giant-cell tumors is available. Janasson treated 4 patients with this lesion using irradiation alone and reported 10- to 12-year cures. His dosage was 500 to 1 000 gamma given every second month until ossification appeared. This took from 3 to 4 months and he states that it was adequate treatment. Roentgenotherapists differ in their opinions on dosage. Some feel that 500 gamma is sufficient, and many feel that more than 3 000 gamma may give rise to an irradiation sarcoma in 5 to 10 years. One fourth of these cases were treated by x rays and were complicated by pathologic fracture. Coley feels that this can be greatly eliminated by adequate splinting.

Surgery consists of curettage, resection of the mass together with a good margin of normal bone or amputation. Tumors occurring in the lower femur or the upper tibia are easily accessible and amenable to curettage. Lesions in the ulna and the fibula, if extensive are amenable to resection. Frankly extensive and malignant giant-cell tumors or recurrent Grade II giant-cell tumors should have amputation.

The combination of surgery and irradiation has not met with good results due to wound breakdown, devitalization of bone and sarcomatous transformation.

PROGNOSIS

Fifty per cent of giant-cell tumors will have a favorable outcome whatever mode of accepted therapy is used. One third are likely to recur and the remaining 15 per cent may be malignant on initial examination (Jaffe).

In general the older the patient, the more uncertain is the outlook. Lesions in bones of the upper extremity tend to have a more favorable prognosis than those of the lower extremity.

The grading of the stroma has afforded a valuable tool in determining the malignancy of the tumor. However even this is not

absolute, and recurrent giant-cell tumors following thorough curettage should be resected or have amputation strongly considered.

SUMMARY

Giant-cell tumor of bone can be a difficult diagnosis to make. A review of the history of this tumor reveals the many controversies concerning its natural history as well as its degree of malignancy.

The pathogenesis most widely accepted is that this is a true tumor of bone arising from the marrow's nonosseous forming connective tissue.

Principal symptoms in this lesion are trauma, pain, tumor and fracture. The greatest age incidence falls between 20 and 50 years. Males are affected about equally with females. The tumor occurs in the ends of long tubular bones and roentgenograms show it as a radiolucent trabeculated mass expanding the epiphyseal end of the bone. Gross examination reveals a bloody necrotic tumor mass. Microscopic examination has been greatly augmented by grading the stroma as described by Jaffe and Lichtenstein. The grading extends from the more benign loose stroma with occasional atypism to the malignant markedly anaplastic stroma.

Since such lesions as aneurysmal bone cyst, nonossifying fibroma, ossifying fibroma, chondroblastoma, chondromyxoid fibroma, and solitary unicameral bone cyst have been confused repeatedly with giant-cell tumor of bone a differential diagnosis of these lesions is presented.

Treatment consists of irradiation, curettage, resection and amputation. Irradiation is capable of controlling the more benign lesions in poorly accessible surgical sites. Lesions in the ulna or the fibula can be resected whereas in the proximal tibia and the distal femur they are readily amenable to curettage. Frankly malignant lesions should be amputated.

Fifty per cent of giant-cell tumors will do well with any type of accepted treatment. One third are apt to recur and about 15 per cent are frankly malignant.

GIANT CELL TUMOR

- ✓Geschickter C. F. Treatment of giant cell tumor of long bones by surgery by irradiation J Bone & Joint Surg. 17 550 1935
- Geschickter C. F., and Copeland M. M. Giant cell tumor and osteitis fibrosa cystica Arch Surg 19 169 1929
- Geschickter C. F. and Widehorn H. L. Giant cell tumors Relationship to histogenesis of osteitis fibrosa cystica, Arch f klin. Chr 172.694 1933
- Jaffe, H. L. Lichtenstein L. and Portus, R. B. Giant cell sarcoma and a new granuloma of bone, M J Australia 1 241 1941
- Giant cell tumor of bone Its pathologic appearance grading, supposed variants and treatment, Arch Path 30 993 1940
- Lichtenstein, L. Giant cell tumor of bone Current status of problems in diagnosis and treatment J Bone & Joint Surg 35-A.143 1951
- Linde, S. A. Giant cell tumor of patella complete review of literature Am J Surg. 28 150 1935
- Mallory F. B. Giant cell sarcoma J M Research 19-363 1911
- Nélaton E. D'une nouvelle espèce de tumeurs bénignes des os, ou tumeurs à myéloplaxes, Paris, Adrien Delahaye, 1860
- Paget Sir J. Lectures on Surgical Pathology (delivered at the Royal College of Surgeons of England) London, Longmans, 1933
- Stewart, M. J. Observations on myeloid sarcoma with an analysis of 50 cases, Lancet 2 1236 1914
- The histogenesis of myeloid sarcoma with a criticism of the "chronic hemorrhage osteomyelitis" theory Lancet 2.1106 1922.
- Stewart, F. W. Coley B. L. and Farrow J. H. Malignant giant cell tumor of bone Am J Path. 14 515 1938
- Bergstrand H. Genesis of giant cell tumors, Am J Cancer 27 701 1936
- Bloodgood J. C. Diagnosis and treatment of benign and malignant tumors of bone J Radiol 1 147 1920
- Benign giant cell tumor of bone Its diagnosis and conservative treatment, Am J Surg. 37 105 1923
- The giant cell tumor of bone and the specter of the metastasizing giant cell tumor Surg., Gynec & Obst 38 784 1924
- Christensen, F. C. Bone tumors. Analysis of one thousand cases with special reference to location, age and sex, Ann. Surg. 81 1074 1925
- Codman E. A. Treatment of giant cell tumors about knee Surg Gynec & Obst. 64 485 1937
- Coley B. L. and Higinbotham N. L. Surgical treatment of giant cell tumor Ann Surg. 103 821 1936
- ✓Giant cell tumor of bone J Bone & Joint Surg 20 870 1938
- Coley B. L., and Miller L. E. Atypical giant cell tumor Am. J Roentgenol 47 541 1942.
- Coley W. B. Prognosis in giant cell sarcoma of the long bones, Ann. Surg. 79 321 1924
- Malignant changes in the so-called benign giant cell tumor Am J Surg 28 768 1935
- Cooper Sir Astley and Travers, B. Surgical Essays, London Cox & Son, and Longmans & Co., 1 195 1818
- Geschickter C. F., and Copeland, M. M. Tumors of giant cell group A pathologic study Arch. Surg 21 145 1930
- Willis, R. A. The pathology of osteoclastoma or giant cell tumor of bone J Bone & Joint Surg. 31B 236 1949

ANEURYSMAL BONE CYST

- Bease, B. E., Jr., Dahlin, D. C., Bruwer A. Svien, H. J. and Ghormley R. K. Aneurysmal bone cyst, Proc Staff Meet. Mayo Clinic 28.249 1953
- Jaffe, H. L. Aneurysmal bone cyst Bull. Hosp Joint Dis 11 3 1950
- Lichtenstein Louis Aneurysmal bone cyst. A pathological entity commonly mistaken for giant cell tumor and occasionally for hemangioma and osteogenic sarcoma, Cancer 3 279 1950
- Mayer L. and Kestler O. C. Aneurysmal bone cyst of spine Bull. Hosp Joint Dis 5 16 1944

NONOSSIFYING FIBROMA OF BONE

- Burman, M. S., and Sinberg, S. E. Solitary anethoma (lipoid granulomatosis) of bone Arch. Surg 37 1017 1938
- Jaffe H. L., and Lichtenstein L. Non-osteogenic fibroma of bone Am J Path 18 205 1942

OSSIFYING FIBROMA OF BONE

- Coley B. L. Neoplasms of Bone and Related Conditions Their Etiology Pathogenesis Diagnosis and Treatment, New York Hoeber 1949
- Geschickter C. F., and Copeland M. M. Tumors of Bone ed. 3 Philadelphia Lippincott, 1949

indications have been stated already. Irradiation is capable of controlling effectively giant-cell tumors of the less aggressive type. No reliable series of radiation therapy to giant-cell tumors is available. Janasson treated 4 patients with this lesion using irradiation alone and reported 10- to 12-year cures. His dosage was 500 to 1,000 gamma given every second month until ossification appeared. This took from 3 to 4 months, and he states that it was adequate treatment. Roentgenotherapists differ in their opinions on dosage. Some feel that 500 gamma is sufficient and many feel that more than 3,000 gamma may give rise to an irradiation sarcoma in 5 to 10 years. One fourth of these cases were treated by x rays and were complicated by pathologic fracture. Coley feels that this can be greatly eliminated by adequate splinting.

Surgery consists of curetment resection of the mass together with a good margin of normal bone or amputation. Tumors occurring in the lower femur or the upper tibia are easily accessible and amenable to curettage. Lesions in the ulna and the fibula if extensive are amenable to resection. Frankly extensive and malignant giant-cell tumors or recurrent Grade II giant-cell tumors should have amputation.

The combination of surgery and irradiation has not met with good results due to wound breakdown, devitalization of bone and sarcomatous transformation.

PROGNOSIS

Fifty per cent of giant-cell tumors will have a favorable outcome whatever mode of accepted therapy is used. One third are likely to recur and the remaining 15 per cent may be malignant on initial examination (Jaffe).

In general the older the patient the more uncertain is the outlook. Lesions in bones of the upper extremity tend to have a more favorable prognosis than those of the lower extremity.

The grading of the stroma has afforded a valuable tool in determining the malignancy of the tumor. However even this is not

absolute and recurrent giant-cell tumors following thorough curettage should be resected or have amputation strongly considered.

SUMMARY

Giant-cell tumor of bone can be a difficult diagnosis to make. A review of the history of this tumor reveals the many controversies concerning its natural history as well as its degree of malignancy.

The pathogenesis most widely accepted is that this is a true tumor of bone arising from the marrow's nonosseous forming connective tissue.

Principal symptoms in this lesion are trauma, pain, tumor and fracture. The greatest age incidence falls between 20 and 50 years. Males are affected about equally with females. The tumor occurs in the ends of long tubular bones and roentgenograms show it as a radiolucent trabeculated mass expanding the epiphyseal end of the bone. Gross examination reveals a bloody necrotic tumor mass. Microscopic examination has been greatly augmented by grading the stroma as described by Jaffe and Lichtenstein. The grading extends from the more benign loose stroma with occasional atypism to the malignant, markedly anaplastic stroma.

Since such lesions as aneurysmal bone cyst, nonossifying fibroma, ossifying fibroma, chondroblastoma, chondromyxoid fibroma, and solitary unicameral bone cyst have been confused repeatedly with giant-cell tumor of bone a differential diagnosis of these lesions is presented.

Treatment consists of irradiation, curettage, resection and amputation. Irradiation is capable of controlling the more benign lesions in poorly accessible surgical sites. Lesions in the ulna or the fibula can be resected, whereas in the proximal tibia and the distal femur they are readily amenable to curettage. Frankly malignant lesions should be amputated.

Fifty per cent of giant-cell tumors will do well with any type of accepted treatment. One third are apt to recur and about 15 per cent are frankly malignant.

BIBLIOGRAPHY

GIANT-CELL TUMOR

- ✓ Geschickter C. F. Treatment of giant cell tumor of long bones by surgery by irradiation *J Bone & Joint Surg* 17:550 1935
- Geschickter C. F., and Copeland, M. M. Giant cell tumor and osteitis fibrosa cystica, *Arch Surg* 19:169 1929
- Geschickter C. F., and Widehorn H. L. Giant cell tumors Relationship to histogenesis of osteitis fibrosa cystica *Arch. f Klin Chir* 172:694 1933
- Jaffe H. L., Lichtenstein, L., and Portis, R. B. Giant cell sarcoma and a new granuloma of bone, *M J Australia* 1:241 1941
- Giant cell tumor of bone Its pathologic appearance grading supposed variants and treatment, *Arch Path.* 30:993 1940
- Lichtenstein, L. Giant cell tumor of bone Current status of problems in diagnosis and treatment, *J Bone & Joint Surg* 35-A:143 1951
- Linde, S. A. Giant cell tumor of patella complete review of literature *Am J Surg* 28:150 1935
- Mallory F. B. Giant cell sarcoma, *J M Research* 19:363 1911
- Nélaton E. D'une nouvelle espèce de tumeurs bénignes des os, ou tumeurs à myéloplaxes, Paris, Adrien Delahaye, 1860
- Paget, Sir J. Lectures on Surgical Pathology (delivered at the Royal College of Surgeons of England) London, Longmans, 1953
- Stewart M. J. Observations on myeloid sarcoma, with an analysis of 50 cases, *Lancet* 2:1236 1914
- The histogenesis of myeloid sarcoma with a criticism of the "chronic hemorrhage osteomyelitis" theory *Lancet* 2:1106 1922
- Stewart, F. W., Coley B. L., and Farrow J. H. Malignant giant cell tumor of bone *Am. J Path.* 14:515 1938
- Bergstrand, H. Genesis of giant cell tumors, *Am. J Cancer* 27:701 1936
- Bloodgood, J. C. Diagnosis and treatment of benign and malignant tumors of bone, *J Radiol* 1:147 1920
- Benign giant cell tumor of bone Its diagnosis and conservative treatment, *Am J Surg* 37:105 1923
- The giant cell tumor of bone and the specter of the metastasizing giant cell tumor *Surg., Gynec & Obst.* 38:784 1924
- Christensen F. C. Bone tumours. Analysis of one thousand cases with special reference to location, age, and sex, *Ann Surg.* 81:1074 1925
- Codman, E. A. Treatment of giant cell tumors about knee *Surg., Gynec & Obst.* 64:485 1937
- Coley B. L. and Higinbotham, N. L. Surgical treatment of giant cell tumor *Ann. Surg* 103:821 1936
- Giant cell tumor of bone *J Bone & Joint Surg* 20:870 1938
- Coley B. L., and Miller L. E. Atypical giant cell tumor *Am J Roentgenol* 47:541 1942
- Coley W. II. Prognosis in giant cell sarcoma of the long bones *Ann Surg.* 79:321 1924
- Malignant changes in the so-called benign giant cell tumor *Am J Surg.* 28:768 1935
- Cooper Sir Astley and Travers II. Surgical Essays, London, Cox & Son and Longmans & Co., 1:195 1818
- Geschickter C. F., and Copeland, M. M. Tumors of giant cell group A pathologic study *Arch. Surg.* 21:145 1930
- Willis, R. A. The pathology of osteoclastoma or giant cell tumor of bone, *J Bone & Joint Surg* 31B:236 1949

ANEURYSMAL BONE CYST

- Besse B. E., Jr., Dahlin, D. C. Bruwer A., Sven, H. J., and Gbormley R. K. Aneurysmal bone cyst, *Proc. Staff Meet., Mayo Clinic* 28:249 1953
- Jaffe, H. L. Aneurysmal bone cyst, *Bull Hosp Joint Dis.* 11:3 1950
- Lichtenstein, Louis. Aneurysmal bone cyst. A pathological entity commonly mistaken for giant cell tumor and occasionally for hemangioma and osteogenic sarcoma, *Cancer* 3:279 1950
- Mayer L., and Kestler O. C. Aneurysmal bone cyst of spine, *Bull. Hosp Joint Dis.* 5:16 1944

NONOSSIFYING FIBROMA OF BONE

- Burman, M. S., and Sinberg, S. E. Solitary anothoma (lipoid granulomatosis) of bone *Arch Surg* 37:1017 1938
- Jaffe H. L., and Lichtenstein L. Non-osteogenic fibroma of bone *Am. J Path.* 18:205 1942.

OSSIFYING FIBROMA OF BONE

- Coley II. L. Neoplasms of Bone and Related Conditions Their Etiology Pathogenesis, Diagnosis and Treatment, New York, Hoeber 1949
- Geschickter C. F., and Copeland M. M. Tumors of Bone ed 3 Philadelphia, Lippincott, 1949

indications have been stated already. Irradiation is capable of controlling effectively giant-cell tumors of the less aggressive type. No reliable series of radiation therapy to giant-cell tumors is available. Janasson treated 4 patients with this lesion using irradiation alone and reported 10- to 12-year cures. His dosage was 500 to 1 000 gamma given every second month until ossification appeared. This took from 3 to 4 months and he states that it was adequate treatment. Roentgenotherapists differ in their opinions on dosage. Some feel that 500 gamma is sufficient, and many feel that more than 3 000 gamma may give rise to an irradiation sarcoma in 5 to 10 years. One fourth of these cases were treated by x rays and were complicated by pathologic fracture. Coley feels that this can be greatly eliminated by adequate splinting.

Surgery consists of curettage, resection of the mass together with a good margin of normal bone or amputation. Tumors occurring in the lower femur or the upper tibia are easily accessible and amenable to curettage. Lesions in the ulna and the fibula, if extensive, are amenable to resection. Frankly extensive and malignant giant-cell tumors or recurrent Grade II giant-cell tumors should have amputation.

The combination of surgery and irradiation has not met with good results due to wound breakdown, devitalization of bone, and sarcomatous transformation.

PROGNOSIS

Fifty per cent of giant-cell tumors will have a favorable outcome whatever mode of accepted therapy is used. One third are likely to recur and the remaining 15 per cent may be malignant on initial examination (Jaffe).

In general the older the patient the more uncertain is the outlook. Lesions in bones of the upper extremity tend to have a more favorable prognosis than those of the lower extremity.

The grading of the stroma has afforded a valuable tool in determining the malignancy of the tumor. However even this is not

absolute, and recurrent giant-cell tumors following thorough curettage should be resected or have amputation strongly considered.

SUMMARY

Giant-cell tumor of bone can be a difficult diagnosis to make. A review of the history of this tumor reveals the many controversies concerning its natural history as well as its degree of malignancy.

The pathogenesis most widely accepted is that this is a true tumor of bone arising from the marrow's nonosseous forming connective tissue.

Principal symptoms in this lesion are trauma, pain, tumor and fracture. The greatest age incidence falls between 20 and 50 years. Males are affected about equally with females. The tumor occurs in the ends of long tubular bones and roentgenograms show it as a radiolucent trabeculated mass expanding the epiphyseal end of the bone. Gross examination reveals a bloody necrotic tumor mass. Microscopic examination has been greatly augmented by grading the stroma as described by Jaffe and Lichtenstein. The grading extends from the more benign loose stroma with occasional atypism to the malignant, markedly anaplastic stroma.

Since such lesions as aneurysmal bone cyst, nonossifying fibroma, ossifying fibroma, chondroblastoma, chondromyxoid fibroma, and solitary unicameral bone cyst have been confused repeatedly with giant-cell tumor of bone a differential diagnosis of these lesions is presented.

Treatment consists of irradiation, curettage, resection and amputation. Irradiation is capable of controlling the more benign lesions in poorly accessible surgical sites. Lesions in the ulna or the fibula can be resected whereas in the proximal tibia and the distal femur they are readily amenable to curettage. Frankly malignant lesions should be amputated.

Fifty per cent of giant-cell tumors will do well with any type of accepted treatment. One third are apt to recur and about 15 per cent are frankly malignant.

upon (1) anatomic structures involved and (2) treatment received.

Sprains may be divided into two large classes (1) articular, or those affecting the joints themselves, and (2) muscular, concerned with overstretching, or dislocation of muscles and their tendons

Articular sprains may be divided into (a) simple sprains and (b) complicated sprains. In the simplest form of sprain that is technically described as a strain the soft parts merely are stretched beyond their capacity, and there is supposedly no laceration. No swelling is to be found and the symptoms are only those of pain and stiffness with some loss of function. In the more severe forms of simple sprains in which there has been more or less tearing of ligamentous structures swelling occurs. Swelling immediately after an injury is due to hemorrhage. Arteries as well as veins are torn and pour their contents into surrounding tissues. Swelling that occurs hours or even days later is due to serous effusion. Delayed swelling in a joint injury in itself produces further symptoms and prolongs recovery. It is not necessary and proper early treatment can circumvent it. For emphasis then joint injuries occur in which swelling is immediate, is generally extensive and is due to hemorrhage. In others pain and limited function are immediate and swelling is delayed for from 12 to 24 hours. In this period of grace something can be done to alleviate the occurrence of secondary effusion and to shorten greatly the period of convalescence. It not actually reduce the likelihood of some degree of permanent disability. Of this I shall talk later.

BRIEF REGIONAL ANALYSES

The most common injury involving the fingers manifests itself in the form of traumatic arthritis with serous or sanguinous effusion. Such injuries are best treated by immobilization in a slightly flexed position for 7 to 10 days. Unfortunately many patients have such injuries brought to a tongue-depressor blade. In healing the extensor sheath or lateral digital

hinges shorten and create a stiff finger which at that point may require many months of further treatment in an effort to restore usefulness. One of my many aphorisms to residents is "The tongue depressor blade is the commonest cause of stiff fingers."

It is indeed a sad commentary on the ability of the average doctor that the Army during World War II found it necessary to issue a directive to the effect that no hand or finger injuries were to be immobilized in the forward units. Apparently an injured finger is not of sufficiently great importance for doctors as a whole to concern themselves to acquire the art of proper immobilization even though all standard textbooks devote considerable space to the discussion of the proper immobilization of the hand and fingers. Our favorite finger dressing is composed of two longitudinal strips of gauze bandage overlaid with several coatings of collodion. This forms a well fitting bandage which protects the injured finger while permitting full use of the hand. A similar dressing may be used for injured toes.

In regard to the wrist joint and the hand one sees few injuries which could be classified as a sprain. Washerwoman's sprain (DeQuervain's Disease—chronic stenosing tendosynovitis) is a chronic constriction of the extensor tendon sheath of the thumb. The principal disability is inability to wring clothes. It is more prevalent in women hence the name. Local infiltration of 0.1 cc of hydrocortisone into the sheath relieves the symptoms dramatically. It may even result in a cure if the patient can be educated sufficiently to avoid all motions comparable with the one used in wringing clothes. If the doctor is not sufficiently expert with a hypodermic needle to locate the swollen extensor sheath easily or if the patient is an over-sensitive subject usually the same result may be obtained by the oral administration of hydrocortisone. The usual oral treatment consists of 4 daily doses of 20 mg. of hydrocortisone for as short a period as 2 days at which time the disability may have cleared up completely. This latter method is a rather

BIBLIOGRAPHY

GIANT-CELL TUMOR

- ✓Geschickter C. F. Treatment of giant cell tumor of long bones by surgery by irradiation, *J Bone & Joint Surg.* 17 550 1935
- Geschickter C. F., and Copeland, M. M. Giant cell tumor and osteitis fibrosa cystica, *Arch Surg.* 19:169 1929
- Geschickter C. F., and Widehorn, H. L. Giant cell tumors Relationship to histogenesis of osteitis fibrosa cystica *Arch. f. klin. Chir.* 172 694 1933
- Jaffe, H. L. Lichtenstein, L., and Portis, R. H. Giant cell sarcoma and a new granuloma of bone *M J Australia* 1 241 1941
- Giant cell tumor of bone Its pathologic appearance grading supposed variants and treatment, *Arch. Path.* 30:993 1940
- Lichtenstein, L. Giant cell tumor of bone. Current status of problems in diagnosis and treatment, *J Bone & Joint Surg.* 33-A:143 1951
- Linde, S. A. Giant cell tumor of patella complete review of literature, *Am J Surg* 28 150 1935
- Mallory F. B. Giant cell sarcoma, *J M. Research* 19 363 1911
- Nélaton, E. D'une nouvelle espèce de tumeurs bénignes des os, ou tumeurs à myélopiloses, Paris, Adrien Delahaye, 1860
- Paget, Sir J. Lectures on Surgical Pathology (delivered at the Royal College of Surgeons of England) London, Longmans, 1953
- Stewart, M. J. Observations on myeloid sarcoma, with an analysis of 50 cases, *Lancet* 2 1236 1914
- The histogenesis of myeloid sarcoma with a criticism of the "chronic hemorrhage osteomyelitis" theory *Lancet* 2:1106 1922
- Stewart, F. W. Coley B. L., and Farrow J. H. Malignant giant cell tumor of bone *Am J Path.* 14:515 1938
- Bergstrand, H. Genesis of giant cell tumors, *Am J Cancer* 27 701 1936
- Bloodgood, J. C. Diagnosis and treatment of benign and malignant tumors of bone, *J Radiol* 1 147 1920
- Benign giant cell tumor of bone Its diagnosis and conservative treatment *Am. J Surg.* 37 105 1923
- The giant cell tumor of bone and the specter of the metastasizing giant cell tumor *Surg., Gynec. & Obst.* 38 784 1924
- Christensen, F. C. Bone tumours. Analysis of one thousand cases with special reference to location, age, and sex, *Ann Surg* 81 1074 1925

- Codman E. A. Treatment of giant cell tumors about knee *Surg Gynec. & Obst.* 64:485 1937
- Coley B. L. and Higinbotham N. L. Surgical treatment of giant cell tumor *Ann. Surg.* 103 821 1936
- Giant cell tumor of bone, *J Bone & Joint Surg.* 20 870 1938
- Coley B. L. and Miller L. E. Atypical giant cell tumor *Am. J Roentgenol.* 47:541 1942
- Coley W. B. Prognosis in giant cell sarcoma of the long bones, *Ann. Surg.* 79 321 1924
- Malignant changes in the so-called benign giant cell tumor *Am. J Surg* 28 768 1935
- Cooper Sir Astley and Travers, B. *Surgical Essays*, London, Cox & Son, and Longmans & Co 1 195 1818
- Geschickter C. F., and Copeland M. M. Tumors of giant cell group A pathologic study *Arch. Surg* 21 145 1930
- Willis, R. A. The pathology of osteoclastoma or giant cell tumor of bone *J Bone & Joint Surg.* 31B 236 1949

ANEURYSMAL BONE CYST

- Besse, B. E. Jr. Dahlin, D. C., Bruwer A. Svien, H. J., and Ghormley R. K. Aneurysmal bone cyst, *Proc. Staff. Meet., Mayo Clinic* 28:249 1953
- Jaffe H. L. Aneurysmal bone cyst, *Bull. Hosp Joint Dis.* 11 3 1950
- Lichtenstein, Louis. Aneurysmal bone cyst. A pathological entity commonly mistaken for giant cell tumor and occasionally for hemangioma and osteogenic sarcoma, *Cancer* 3:279 1950
- Mayer L., and Keatler O. C. Aneurysmal bone cyst of spine *Bull. Hosp Joint Dis.* 5 16 1944

NONOSSIFYING FIBROMA OF BONE

- Burman M. S., and Sinberg S. E. Solitary anothoma (lipoid granulomatosis) of bone *Arch. Surg* 37 1017 1938
- Jaffe H. L., and Lichtenstein, L. Non-osteogenic fibroma of bone, *Am. J Path.* 18:205 1942.

OSSIFYING FIBROMA OF BONE

- Coley B. L. Neoplasms of Bone and Related Conditions Their Etiology Pathogenesis Diagnosis, and Treatment, New York, Hoeber 1949
- Geschickter C. F., and Copeland, M. M. Tumors of Bone, ed. 3 Philadelphia, Lippincott, 1949

Jaffe H. L., and Mayer L. An osteoblastic osteoid tissue forming tumor of a metacarpal bone Arch Surg 24 550 1932

CHONDROMYXOID FIBROMA OF BONE

Bloodgood, J. C. Bone tumors myxoma, Ann Surg. 80 817 1924

Jaffe H. L., and Lichtenstein L. Chondromyxoid fibroma of bone = distinctive benign tumor likely to be mistaken especially for chondrosarcoma, Arch. Path. 45:541 1948

Lichtenstein, L. Chondromyxoid fibroma of bone (abstract.) Am J Path. 24 686 1948

Stradford H. T. Chondromyxoid fibroma of bone, Bull. Charlotte Mem Hosp 3 7 1948

Willis, R. A. Pathology of Tumors, p 684 and Fibroma 329 London Butterworth, 1948

BENIGN CHONDROBLASTOMA OF BONE

Codman E. A. Epiphyseal chondromatous giant cell tumors of the upper end of the humerus, Surg. Gynec. & Obst. 52:543 1931

Coley B. L., and Santoro A. J. Benign central cartilaginous tumors of bone Surgery 22:411 1947

Geschickter C. F. Recurrent and so-called metastatic giant cell tumor Arch Surg. 20 715 1930

Geschickter C. F. and Copeland, M. M. Osteitis fibrosa and giant cell tumor Arch. Surg 19 169 1929

—— Chondroblastic tumors of bone benign and malignant Ann Surg 129:724 1949

Jaffe H. L., and Lichtenstein, L. Benign chondroblastoma of bone, Am J Path 18 969 1942.

Kolodny A. Bone sarcoma primary malignant tumors of bone and giant cell tumor Surg. Gynec. & Obst 4:1 1927

Lichtenstein L., and Kaplan, L. Benign chondroblastoma of bone unusual localization in femoral capital epiphysis Cancer 2:793 1949

SOLITARY (UNICAMERAL) BONE CYST

Albright, F. Butler A. M., Hampton A. O., and Smith P. Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction with precocious puberty in females. Report of five cases, New England J Med. 216 727 1947

Bennett, C. B. Notes on early bone cyst, Arch. Surg. 46 608 1943

Bloodgood, J. C. Benign bone cyst, osteitis fibrosa, giant cell sarcoma and bone aneurysm of the long pipe bones. A clinical and pathological study with the conclusion that conservative treatment is justifiable Ann Surg. 52:145 1910

Coley B. L., and Higinbotham N. L. Solitary Bone Cyst, Ann Surg. 99:432 1934

Geschickter C. F. and Copeland, M. M. Recurrent and so-called metastatic giant cell tumor Arch. Surg. 20:713 1930

Jaffe, H. L. and Lichtenstein, L. Solitary unicameral bone cyst, with emphasis on the x-ray picture, pathology and pathogenesis, Arch. Surg. 44 1004 1942.

James, A. G., Coley B. L. and Higinbotham N. L. Solitary (unicameral) bone cyst, Arch. Surg. 67 137 1948

Phemister D. B., and Gordon, J. E. The etiology of solitary bone cyst, J.A.M.A. 87 1429 1926

FIBROUS DYSPLASIA OF BONE

Albright, F. Scoville, W. B., and Sulkowitch, H. W. Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation, and gonadal dysfunction further observation with report of 2 more cases Endocrinology 22:411 1938

Falconer M. A., Cope, C. L. and Robb-Smith, A. H. T. Fibrous dysplasia of bone with endocrine disorders and cutaneous pigmentation (Albright's disease) Quart. J. Med. 11 121 1942

Hatcher C. H. The pathogenesis of localized fibrous lesions in the metaphysis of long bones Ann Surg. 122:1016 1945

Jaffe H. L. Fibrous dysplasia of bone Bull. New York Acad. Med. 22:588 1946.

Lichtenstein L., and Jaffe H. L. Fibrous dysplasia of bone a condition affecting one, several, or many bones, the graver cases of which may present abnormal pigmentation of skin premature sexual development, hyperthyroidism, or still other extra-skeletal abnormalities, Arch. Path. 33 777 1942.

Schlumberger H. G. Fibrous dysplasia of single bones (monostotic fibrous dysplasia) Mil. Surgeon 99:504 1946

Step-Cut Knee Fusion with Medullary Fixation

WM MINOR DEYERLE, M D, and
VIRGIL R. MAY, JR, M D

This arthrodesis employs the established principles of absolute immobilization and contact compression of raw bone surfaces.

Dr J. A. Key¹ in 1932 first advocated positive pressure in knee fusions and accomplished this by inserting a Steinmann pin above and below the knee joint. These pins were put on tension by applying turn buckles which were tightened at intervals. Additional immobilization was obtained by incorporating the pins in a long leg cast including the foot.

Charnley⁴ in 1948 modified this technic by substituting a Thomas splint for the cast in the first 4 weeks of treatment. All 15 of his cases fused in spite of the pins breaking in 4 instances. Fetti⁶ in 1953 modified the procedure further by placing 2 pins above and 2 below the knee in order to give better immobilization in the anterior posterior plane as well as in the lateral plane. He also stated that there was practically no likelihood of fracture of the pins. All of these methods required 4 weeks in bed and at least 8 weeks before weight bearing could be started.

Chapchal² in 1948 reported 7 knees successfully fused using medullary fixation. Stack³ in 1952 reported medullary fixation in 20 knees, including 6 tuberculous knees and 8 Charcot knee joints. Only one of the Charcot knees was fused successfully. All of the others fused in spite of complications as follows: 2 nails broke and the femur fractured at the site of the entrance of the

nail in 2 cases. There was one fracture of the tibia at the distal end of the nail.

My associate Dr May used medullary nails in 2 knee fusions. One patient had an argument with a friend and as you can see lost the argument. (Fig 1.)

Case 1. S.S. 28 year-old colored female, developed a septic knee and osteomyelitis of the compound fracture of the upper end of the tibia. Her knee drained 14 months following the wound but had been healed 5 months prior to surgery. The tibia had united in a grossly angulated position, and the patient could bear no weight on the extremity and had only a few degrees of painful knee motion. The knee joint was denuded and cut in such a manner as to correct the angulation in the lateral plane and fused in approximately 15° flexion, using a 38 cm. x 10 mm. medullary nail. A plaster cast was used from the groin to the ankle and the patient placed on antibiotics. She was discharged from the hospital in 14 days with partial weight bearing on crutches. She had full weight-bearing in 1 month, and all casts were removed in 6 months, at which time there was a solid union (Fig. 2). This woman works daily as a housewife.

Case 2. R.J., 50-year-old colored male, disabled 13 years with a Charcot knee. He was unable to bear weight and walked on crutches (Fig 3). This patient had a similar fusion using a pin 38 cm. x 9 mm. and was walking on crutches in 14 days with partial weight bearing. He left the hospital in 3

- Jaffe, H. L. and Mayer, L. An osteoblastic osteoid tissue forming tumor of a metacarpal bone Arch. Surg. 24:550 1932
- CHONDROMYXOID FIBROMA OF BONE
- Bloodgood, J. C. Bone tumors myxoma, Ann. Surg. 80:817 1924
- Jaffe, H. L. and Lichtenstein, L. Chondromyxoid fibroma of bone a distinctive benign tumor likely to be mistaken especially for chondrosarcoma Arch. Path. 45:541 1948
- Lichtenstein, L. Chondromyxoid fibroma of bone (abstract). Am. J. Path. 24:686 1948
- Stratford, H. T. Chondromyxoid fibroma of bone, Bull. Charlotte Mem. Hosp. 3:7 1948
- Willis, R. A. Pathology of Tumors, p 684 and Fibroma 329 London, Butterworth 1948
- BENIGN CHONDROBLASTOMA OF BONE
- Codman, E. A. Epiphyseal chondromatous giant cell tumors of the upper end of the humerus, Surg., Gynec. & Obst. 52:543 1931
- Coley II L. and Santoro, A. J. Benign central cartilaginous tumors of bone, Surgery 22:411 1947
- Geachlacker, C. F. Recurrent and so-called metastatic giant cell tumor Arch. Surg. 20:715 1930
- Geachlacker, C. F., and Copeland, M. Osteitis fibrosa and giant cell tumor Arch. Surg. 19:169 1929
- Chondroblastic tumors of bone benign and malignant, Ann. Surg. 129:724 1949
- Jaffe, H. L. and Lichtenstein, L. Benign chondroblastoma of bone Am. J. Path. 18:969 1942
- Kolodny, A. Bone sarcoma primary malignant tumors of bone and giant cell tumor Surg. Gynec. & Obst. 44:1 1927
- Lichtenstein, L. and Kaplan, L. Benign chondroblastoma of bone unusual localization in femoral capital epiphysis, Cancer 2:793 1949
- SOLITARY (UNICAMERAL) BONE CYST
- Albright, F. Butler, A. M., Hampton, A. O., and Smith, P. Syndrome characterized by osteitis fibrosa diseminata, areas of pigmentary and endocrine dysfunction, with precocious puberty in females. Report of five cases, New England J. Med. 216:727 1947
- FIBROUS DYSPLASIA OF BONE
- Albright, F. Scoville, W. B. and Sulkowitch, H. W. Syndrome characterized by osteitis fibrosa diseminata, areas of pigmentary, and gonadal dysfunction further observation with report of 2 more cases, Endocrinology 22:411 1938
- Falconer, M. A. Cope, C. L. and Robt-Smith, A. H. T. Fibrous dysplasia of bone with endocrine disorders and cutaneous pigmentation (Albright's disease) Quart. J. Med. 11:121 1942
- Hatcher, C. H. The pathogenesis of localized fibrous lesions in the metaphysis of long bones, Ann. Surg. 122:1016 1945
- Jaffe, H. L. Fibrous dysplasia of bone, Bull. New York Acad. Med. 22:588 1946
- Lichtenstein, L. and Jaffe, H. L. Fibrous dysplasia of bone a condition affecting one, several, or many bones, the greater cases of which present abnormal pigmentation of skin, precocious sexual development, hyperthyroidism, or still other extra-skeletal abnormalities, Arch. Path. 33:777 1942
- Schulmberger, H. O. Fibrous dysplasia of single bones (monostotic fibrous dysplasia) N. Engl. Surgon 99:504 1946

Step-Cut Knee Fusion with Medullary Fixation

WM MINOR DEYERLE, M.D., and
VIRGIL R. MAY, JR. M.D.

This arthrodesis employs the established principles of absolute immobilization and contact compression of raw bone surfaces.

Dr J A Key¹ in 1932 first advocated positive pressure in knee fusions and accomplished this by inserting a Steinmann pin above and below the knee joint. These pins were put on tension by applying turn buckles which were tightened at intervals. Additional immobilization was obtained by incorporating the pins in a long leg cast including the foot.

Charnley⁴ in 1948 modified this technique by substituting a Thomas splint for the cast in the first 4 weeks of treatment. All 15 of his cases fused in spite of the pins breaking in 4 instances. Feit⁵ in 1953 modified the procedure further by placing 2 pins above and 2 below the knee in order to give better immobilization in the anterior posterior plane, as well as in the lateral plane. He also stated that there was practically no likelihood of fracture of the pins. All of these methods required 4 weeks in bed and at least 8 weeks before weight bearing could be started.

Chapchal in 1948 reported 7 knees successfully fused using medullary fixation. Stack² in 1952 reported medullary fixation in 20 knees, including 6 tuberculous knees and 8 Charcot knee joints. Only one of the Charcot knees was fused successfully. All of the others fused in spite of complications as follows: 2 nails broke, and the femur fractured at the site of the entrance of the

nail in 2 cases. There was one fracture of the tibia at the distal end of the nail.

My associate, Dr May used medullary nails in 2 knee fusions. One patient had an argument with a friend and, as you can see lost the argument. (Fig. 1)

Case 1. S.S. 28 year-old colored female developed a septic knee and osteomyelitis of the compound fracture of the upper end of the tibia. Her knee drained 14 months following the wound but had been healed 5 months prior to surgery. The tibia had united in a grossly angulated position and the patient could bear no weight on the extremity and had only a few degrees of painful knee motion. The knee joint was denuded and cut in such a manner as to correct the angulation in the lateral plane and fused in approximately 15° flexion, using a 38 cm x 10 mm. medullary nail. A plaster cast was used from the groin to the ankle and the patient placed on antibiotics. She was discharged from the hospital in 14 days with partial weight bearing on crutches. She had full weight bearing in 1 month, and all casts were removed in 6 months, at which time there was a solid union (Fig. 2). This woman works daily as a housewife.

Case 2. R.J. 50-year-old colored male disabled 13 years with a Charcot knee. He was unable to bear weight and walked on crutches (Fig. 3). This patient had a similar fusion using a pin 38 cm. x 9 mm. and was walking on crutches in 14 days with partial weight-bearing. He left the hospital in 3



Fig. 1 Old septic knee. Malalignment of tibia with osteomyelitis following gunshot wound (May V R. South. M J 47 32)



Fig. 2 Septic knee 6 months after arthrodesis. (May V R. South M J 47 32)

area of bone contact by the size of the step-cut. It is technically simple and makes possible early ambulation and weight bearing. The procedure was carried out on post-mortem specimens and gave such excellent immobilization that I did not believe that any external cast would be necessary. I advocate the use of a cast from ankle to groin, because it gives added immobilization, especially above and below the pin and has no disadvantages.

OPERATIVE TECHNIC

If a tourniquet is used, it must be applied very high and held up by several gauze loops or an Esmarch tourniquet may be applied in the sterile field between the two incisions. I prefer not to use a tourniquet as it makes insertion of the pin in the middle third and lower third technically more difficult. Others² have advocated a long median incision inserting the nail from the top portion of the incision that exposes the knee. This requires more surgery and gives less exposure than the U-incision over the

involved. This procedure also increases the logic forces of the musculature of the parts ideal pressure is that exerted by the physio-osteo-genesis. As Eggers³ pointed out, the sion principle with its proved stimulus to placed without loss of the contact compres-nail. This fixation of rotation is accom-absorption of bone around the medullary knee joint could prevent rotation even after $\frac{1}{4}$ inch step-cut in the sagittal plane of the. It seemed to be a logical conclusion that at the fracture site to prevent this rotation. bones I have used a short $\frac{1}{4}$ inch step-cut several occasions in fractures of the long in many cases continues for months. On starting the first few days after insertion and especially true after absorption of bone around the pin, which takes place gradually less effective in preventing rotation. This is mobilization against lateral stresses but is A medullary nail alone gives excellent im steadily since then at heavy labor solid knee fusion (Fig. 4). He has worked and he had clinical and x ray evidence of a months. His cast was removed in 6 months, weeks and had full weight bearing in 2

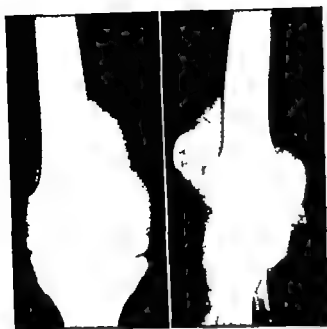


FIG. 3 Charcot knee Disabled 13 years (May V R. South. M J 47 32)



FIG. 4 Charcot knee 6 months after arthrodesis. (May V R. South M J 47 32)

knee and a separate 4-inch incision over the medial portion of the femur. The knee joint is exposed and denuded of all soft tissue. With a broad osteotome the ends are fashioned into a sagittal step-cut pattern $\frac{1}{4}$ inch deep (Fig 5).

The procedure may be simplified by step-cutting the femoral fragment initially and step-cutting the tibial portion only after the nail has been driven down into the femoral fragment flush with the step-cut end of the femur. In this way one can step-cut the tibia in such a fashion as to direct the nail directly into the center portion of the medullary cavity and give maximum apposition of all surfaces.

The femur is exposed through the anterior 4-inch incision at approximately the juncture of the middle and the lower thirds. From 7 to 8 inches above the knee a slot 1 x 4 cm. is cut carefully with multiple drill holes connected by an osteotome or with a power saw. A nail of the proper size is selected, usually 38 to 42 cm. long and 9 or 10 mm. in diameter.

It is inserted carefully into the preformed slot, and if any difficulty is encountered, one should not hesitate to make the slot longer. If the cutting of the slot and the inserting of

the nail are performed carefully most of the fractures at this point can be avoided. The nail is driven down until it is flush with the knee joint. With the step-cut edges held tightly together at 10° to 15° flexion, the

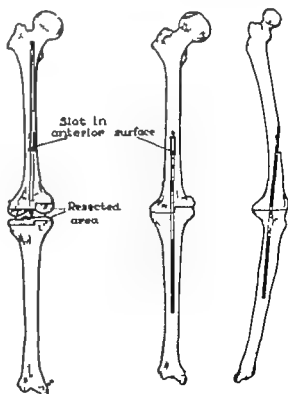


FIG. 5 Step-cut procedure (May V R. South M J 47 32)



FIG 6 Tuberculous knee (May V R. South, M J 47 32)



FIG 7 Tuberculous knee 6 months after step-cut fusion (May V R South, M J 47 32)



stability. If the nail becomes embedded solidly in the tibia or tends to distract the joint, obtain roentgenograms and if everything is satisfactory the excess of the nail can be cut off just above the slot. This leaves a firmly fixed knee. The wounds are closed in the routine manner and a long leg cylinder cast is applied from the ankle to the groin. If it is driven into the slot there is less

CASE REPORT

A W G 28-year-old white male, admitted to the hospital June 9, 1952. His chief complaint was pain, swelling and progressive stiffness of the left knee joint.

Past History In 1944 the patient had a medial meniscectomy performed in the Navy for instability of the left knee. Following this he developed pain and swelling and never recovered completely. In 1945 he was discharged with 20 per cent general disability. In 1946 the patient was hospitalized for his progressing disability. A complete synovectomy was done through 2 parapatella incisions. Frozen sections were done at the time as tuberculosis was suspected. Preoperative and postoperative diagnosis was chronic synovitis. On admission his chest roentgenograms revealed an old fibrotic healed tubercular lesion in his chest. After operation he was discharged from the hospital with full extension and 30° flexion with good lateral stability.

Present Illness. Complete history at the time of his recent admission was negative with the exception of the complaints referable to the left lower extremity. There had been no weight loss, night sweats, cough or generalized weakness. Physical examination was normal except for the left lower extremity. There were well-healed medial and lateral parapatella scars. The left knee was considerably enlarged and endured with a moderate joint effusion. There was some tenderness over the entire joint space, and motion was restricted from 170° to 150° the patient having lost 10° extension since his discharge following synovectomy. There was marked atrophy of the quadriceps muscle.

Roentgenograms. Films of the left knee showed chronic arthritis consistent with tuberculosis (Fig. 6). Roentgenograms of the chest revealed old fibrotic infiltration of the left upper lung unchanged from that noted in 1946.

Laboratory Studies. These were within normal limits, except the white blood count of 11 900. 86 per cent polymorphonuclear

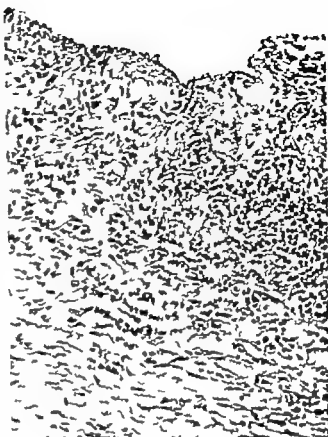


FIG. 8 Chronic synovitis removed from knee in 1947

leukocytes sedimentation rate 34 mm. The patient was afebrile, and the pulse was normal. Sputum smears were negative.

Consultation. The medical service was called in regarding pulmonary tuberculosis and they felt that it was inactive and no contraindication to surgery.

Operative Report. June 23, 1952. Through a 5 inch U flap incision the distal end of the femur and the proximal end of the tibia were completely denuded. The cartilage was moderately destroyed over the tibia and the femur. There was some panus formation and injection of the synovium. There were areas of necrosis on both the femur and the tibia immediately underlying the cartilage. The end of the tibia and the femur were step-cut in a sagittal plane with the step being approximately 3/4 inch deep (Fig. 5). A cloverleaf medullary nail, 9 x 42 mm. was driven into a preformed slot

eventual, although the patient ran a fever up to 100° for approximately 10 days. In 18 days the sutures were removed. The wound was healed per primam. A new cast was applied from the groin to the ankle. The patient was allowed to walk, bearing weight on the limb and using crutches to steady his gait. In 2 months he began almost full weight bearing, using one crutch for support. In 3½ months all casts and supports were removed and there was clinical union. Roentgenograms revealed almost complete obliteration of the joint line. The patient has walked asymptotomically since that time and has a solidly fused knee (Fig 7). Measure- ments reveal ¾ inch shortening of the left leg. Sputum cultures taken prior to opera- tion and reported 60 days later revealed evi- dence of pulmonary tuberculosis. Material taken at surgery from the knee joint revealed tubercular granulation tissue and cultures taken from this showed tubercular bacilli. The patient was turned over to the medical department for care of the pulmonary lesion. A body section roentgenogram September 11, 1952 revealed definite cavitation in the right apex. The same pathologist reviewed the material removed at the time of synovec- tomy and compared it with the material re- moved at the time of the knee fusion and is still unable to make a diagnosis of tubercu- losis on the basis of the initial synovectomy (Figs 8 and 9).

Possible Complications. Although Doc- tor May and I have encountered no compli- cations in our three cases I feel that when one advocates a procedure one also should have a plan to cope with any possible com- plications reported by others.

1. FRACTURE AT THE SITE OF THE INSER- TION OF THE NAIL. If this occurs at the time of operation, remove the pins and insert a long 65-cm nail down by way of the tro- chanter across the fracture site and into the tibia. This has the slight disadvantage of a straight knee. If the fracture occurs after roentgenograms show that union is pro- gressing I suggest pulling a Kirschner wire through the femoral condyles just superior

needed. The postoperative course was un- for pain. After 4 days only aspirin was operatively the usual sedatives were given salicylic acid 4 Gm 3 times a day. Post- streptomycin 1 Gm a day and para amino- prior to surgery the patient was started on fact that tuberculosis was suspected. 110 days Postoperative Progress. In view of the over sheathing from ankle to groin. plain to the skin. A long leg cast was applied No 0 plain catgut subcutaneously and No 0 incision of the anterior thigh was closed with and silk to close the skin edges. The 3-inch make the margins of the subcutaneous tissue No 0 plain catgut was employed to approxi- 15° flexion. The wound was closed in layers good contact of all bony surfaces in about the knee joint into the tibia. Excellent im- the lower thirds of the femur down across 1 x 5 cm at the junction of the middle and



FIG 9 Tubercle in bone removed from knee at fusion in 1952



FIG 1 (Left) Murphy's sign for fractured navicular

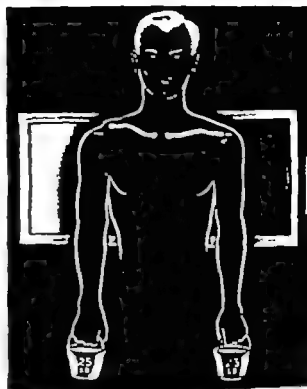


FIG 2 (Right) Proper roentgenogram technique for demonstrating acromioclavicular separation

more expensive one than the use of local injection. It must be anticipated that in a certain percentage of cases the patients are constitutionally or economically unable to eliminate the precipitating motion, i.e. the one used in wringing clothes, and so will have a recurrence of the stenosing tendosynovitis. Under such conditions repeated hydrocortisone therapy may become impractical. Excellent results have been obtained in the past by surgical release of the constricted sheath. It is probable that some cases will not respond to hydrocortisone therapy and will still require surgery.

The common wrist sprain is really a fractured navicular. All are aware that making a roentgenogram of the navicular bone is technically difficult, and that the fracture line may be obscured in the initial roentgenograms. In fact, in a large Army series 40 per cent of fractured naviculars were missed on the initial roentgen ray examination so it is important to remember in wrist injuries that are not responding to treatment, to roentgenograph them again in from 7 to 10 days at which time it is more likely that the fracture line will be apparent.

Clinically it is not necessary to have a roentgenogram to diagnose a fractured navicular.

As Murphy's sign will do it, Murphy's sign (Fig. 1) consists of pounding on the knuckle of the index metacarpal bone with the wrist in neutral position.⁸ If a fractured navicular is present pain will be more pronounced when pounding on the index ray. If a lunate dislocation is present, pain will be more pronounced on the middle ray. If Murphy's sign is present for a fractured navicular and the roentgen ray report is negative that wrist should be treated as a fractured navicular that is put in a cast for 10 to 14 days and then again roentgenographed. Similarly sprained thumbs, particularly from fighting incidents or changing tires when the thumb slips and is caught with a driving force on the bent distal phalanx, are not sprains but fractures of the base of the first metacarpal. All of these hazards of course can be avoided by good roentgenograms, and yet roentgen-ray work has its limitations also.

A roentgen ray machine cannot be blamed for failing to diagnose a fracture of the radial head which produces limited wrist motion when the lazy or overworked surgeon or a careless technician included only the wrist joint in the roentgen-ray study. The roentgen-

or posterior to the pin and apply traction. If the knee is solid, remove the nail from the knee and insert a regular femoral nail from the trochanteric region down to treat the fracture of the femur. In all cases, reapply a cylinder cast from toes to groin to help in total immobilization.

2 FRACTURE OF THE NAIL AT THE KNEE

The nail may be removed from the tibia by cutting a slot in the tibia large enough to insert a punch. The nail is tapped into the knee joint and both pieces can be removed. The proximal portion of the nail is removed in the femur and a new and longer nail is inserted. If necessary it should be a size smaller so that it can be extended beyond the weakened portion of the tibia. If union is in progress, treat the knee fusion as indicated with casts, spicas or other immobilization.

3 FRACTURES OF THE TIBIA BELOW THE PIN

Remove the nail and insert a longer and smaller nail across the fracture site then apply a long leg cast.

If these complications take place they will alter the postoperative care according to the necessities in each case.

Conclusions. This knee arthrodesis combines contact compression and immobilization in a manner that allows early weight-bearing. The procedure is simple and can be used in all conditions of the knee requiring fusion.

REFERENCES

- 1 Key J A. Positive pressure in arthrodesis for tuberculous of the knee joint. *South M J* 25:904 1932.
- 2 Chapchal George. Intramedullary pinning for arthrodesis of the knee joint, *J Bone & Joint Surg* 30A:728 1948.
- 3 Stack James H. Experiences with intramedullary fixation in knee fusion. *Am J Surg* 83:291 1952.
- 4 Charley J C. Positive pressure in arthrodesis of the knee joint, *J Bone & Joint Surg* 30B:478 1948.
- 5 Eggers, G W N., Shandler T J and Pomerat C. M. Influence of contact-compression factor on osteogenesis in surgical fractures, *J Bone & Joint Surg* 31A:693 1949.
- 6 Fett, C. and Zorn E. L. Compression arthrodesis of the knee. *J Bone & Joint Surg* 35A:172 1953.

Closed Medullary Pinning of Colles' Fracture

LESLIE V RUSH, M D *

Our first attempt to pin a Colles fracture was made in 1948. This was a routine Colles fracture in a 51 year-old male. The operation was carried out, with the patient's consent, purely as a clinical experiment and without any great expectation of accomplishment. The pin used was of the same design as the present Rush pin, with the exception that it was made of Type 410 stainless steel because at that time Type 316 stainless steel could not be secured in a satisfactory temper.

In this instance there was fairly marked silver fork deformity with anterior luxation of the distal end of the ulna. Reduction was accomplished, and with the hand and the wrist in the Cotton-Loder position the tip of the styloid process of the radius was palpated, and a very small stab wound was made in the skin over this point in the anatomic snuff box. A small drill hole was made near the tip of the styloid process and was converted into an opening which approached the longitudinal axis of the radius. A pin 5 inches long and $\frac{3}{32}$ inch in diameter was then introduced through this opening and driven upward into the medullary cavity of the radius. The pin traveled easily through the cancellous bone and resistance was felt as the sled-runner point engaged the medial wall of the shaft of the radius. As the pin was driven, the proximal or head end could be seen to swing medially exerting pressure against the styloid fragment in

the medial direction. When checkup roentgenograms were made it was found that the radius had been completely reconstructed, and the fracture line was scarcely discernable. To our very pleasant surprise it was then determined that the radius itself was completely stable and that the styloid end of the ulna was in proper relationship to the radius and was stable in this position without tendency to anterior luxation. It was noted further that full range of motion was possible in every direction. Plaster splints were not applied, and the patient was permitted immediate function.

This patient suffered the usual sprain symptoms for 2 or 3 weeks but never lost the ability to make a tight fist or to pronate the forearm. Because of soreness, there was slight limitation of supination and extremes of flexion for about 3 weeks. At the end of 4 weeks the patient had apparently made full recovery without pain or soreness and the fracture line was no longer demonstrable in the x ray film.

In this particular case, although the pin is of undesirable metal alloy being Martensitic and subject to possible corrosion, the pin has not been removed and has given rise to no bone irritation.

In September 1949 this technic was reported (with considerable difficulty because several medical editors felt that intramedullary fixation was a radical procedure) and the study of 6 cases was reviewed. In this report was included the case of an 87 year old woman who received a fragmented im-

*Department of Surgery Rush Memorial Hospital Meridian, Miss.

pacted Colles fracture of the left wrist. This fracture was treated in the same manner and without splints. It healed without deformity. Function never was lost except for slight limitation of full supination and extreme dorsiflexion because of soreness. Three weeks from the date of her injury this little lady combed and arranged her own hair using the injured wrist.

INDICATIONS

In the 1949 report, which expresses our present opinion in this regard, indications were discussed as follows:

Due to the wide variety of so-called Colles fractures it is difficult at this time to draw accurate conclusions as to which specific case is best benefited by pinning. In children the method is probably never indicated because it is not wise unnecessarily to cross an epiphyseal line and because children rarely develop stiffness of the hand and wrist from injury.

Due to the simplicity of the procedure, we feel that it might be indicated in any adult in whom the styloid fragment of the radius is of sufficient size to be grasped satisfactorily by the

head of the pin. In crush-type fractures it appears to be of inestimable value to maintain the proper length and relationship of the styloid end of the radius. In these cases it might be necessary to apply plaster splints at least during a portion of the healing period.

At first glimpse this appears to be a procedure requiring a great deal of skill on the part of the surgeon. Our experience does not lead us to believe that this is true, but it must be borne in mind that a Colles fracture can be an extremely complicated affair and to be treated properly by any means requires a thorough understanding of its mechanics on the part of the surgeon.

Because of the simplicity of its application and the immediate freedom of function that is permitted the extremity we believe the method to be indicated in all adults, and most particularly the aged, in whom a satisfactory fixation can be secured.¹

In October 1952 a critical review of 50 consecutive cases of closed pinning of fractures of the wrist was made. The conclusions of this study are the main basis of this report. Thirty nine additional cases of Colles fractures have been pinned, with comparable results.



FIG. 7 Showing typical fixation Colles fracture with intramedullary pin. This case was an elderly woman.

TIME OF OPERATION

Early cases were all pinned as emergency procedures because it was felt that swelling would obscure accurate palpation of the styloid end of the radius and complicate the procedure. The operation is now deferred from 2 to 4 days for several reasons.

Sprain symptoms are usually severe for the first several days after a Colles fracture, and the patient gets a great deal of comfort from proper splinting. As a temporary treatment, without anesthesia, gentle traction is exerted upon the index and the middle fingers anterior and posterior plaster splints



FIG. 2. A woman 91 years old who had a fracture of the right hip. These photographs were taken postoperative day 1. The right wrist.

of the right
made on the sixth
recently pinned

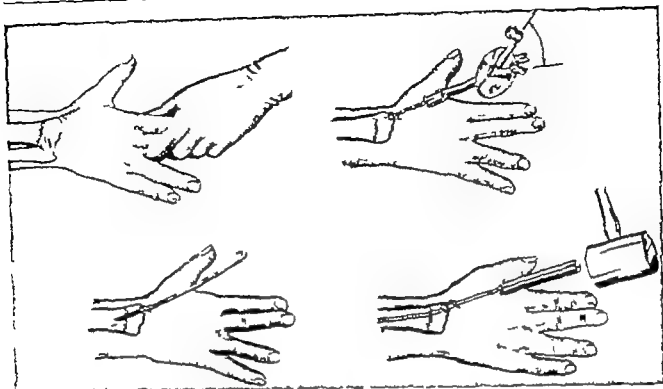


FIG 3 Technic of closed pinning of Colles fracture

are molded in position and held by one or two thicknesses of elastic bandage. This dressing rarely has to be loosened, because it will stretch sufficiently to allow for the swelling that occurs and become snug again as the swelling subsides. The operation can be set at a later date as an elective procedure, which is much more convenient to the surgeon and the staff of the operating room and permits adequate preoperative preparation of the patient.

An additional psychological advantage is gained by delay. When an operation is done immediately the patient is prone to blame all pain and soreness upon the surgeon, losing sight of the fact that he himself suffered an injury prior to his surgery. Also the patient becomes afraid to engage in active motion because of fear of pain.

In delayed operation the patient, recovering from his sprain symptoms, is glad to be rid of his dressing and has an understanding of and confidence in his internal fixation and is more willing to embark upon immediate active motion.

RECONSTRUCTION

The pin chosen is from $4\frac{1}{2}$ to 6 inches in length, there being no need for a pin

longer than 6 inches. Except in rare instances the pin of $\frac{1}{8}$ inch diameter is chosen because the medullary cavity is usually large enough to receive a pin of this diameter. Particularly is this true in the aged in whom the medullary cavity of the long bones is nearly always large. A rare individual has a small medullary cavity in the radius and this may require a pin of $\frac{3}{16}$ inch in diameter. The operation is always done as a closed procedure.

The extremity is prepared from the elbow to the fingertips. The hand and the forearm are passed through an opening in an ordinary laparotomy sheet which is clamped snugly about the mid-portion of the forearm. The fracture is reduced and held in position by straight traction on the middle and the index fingers with the wrist in the neutral position.

Previously the Cotton-Loder position was used for pinning but it was abandoned (1) because it was difficult to palpate the styloid end of the radius (2) because of the tendency to dorsal angulation and (3) because of the possibility of injuring the extensor tendon of the thumb.

Traction widens the gap in the snuffbox between the styloid of

and the base of the thumb so that the tip of the styloid can be palpated even after considerable swelling has occurred. Beginning at the tip of the styloid a $\frac{1}{4}$ inch incision is made through the skin only. This safeguards against injury to tendons, nerves or blood vessels. This incision is made very slightly toward the dorsum about midway between the abductor and the extensor tendons of the thumb. An opening is then made in the styloid end of the radius as near the articular margin as possible passing the instrument through the stab wound. This opening may be made by a drill, an icepick, an awl or as we now prefer by an instrument designed for this purpose which is called the awl reamer. The instrument is first held vertically to the cortex of the bone and rotated back and forth on its axis so as not to entangle soft tissue and is passed just

deeply enough to penetrate the cortex of the bone. Continuing this back and forth rotary motion, gradually the instrument is brought into fairly close alignment with the long axis of the radius and when this position is reached penetration of the bone is increased.

Then the pin is introduced through this opening and pushed up into the medullary cavity of the radius. In the aged and debilitated the cancellous bone is friable and sometimes almost absent, so that the pin can be pushed in with the hand. Yet fixation is usually stable.

In young individuals the cancellous bone is firm and dense and it becomes extremely necessary that the original opening in the bone be made close to the line of the long axis of the bone because if it is made obliquely the distal fragment will be forced to angulate by the passage of the pin, thus

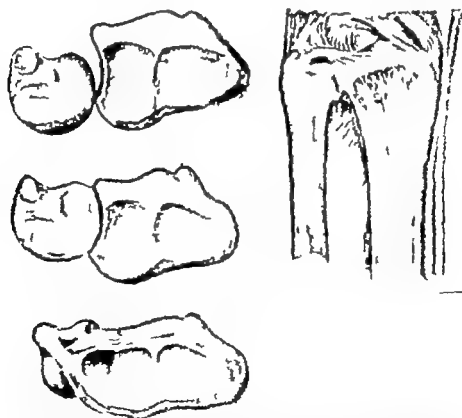


FIG. 4 Drawings showing important radio-ulnar relationship (A Left top) View from the wrist joint to demonstrate anterior luxation of the styloid end of the ulna (B Left center) Normal relationship established. (C Left bottom) Same view showing triangular ligament attachment to tip of styloid (D Right) Dorsal view showing radio-ulnar relationship

giving rise to deformity of the lower end of the radius and a marked disturbance of the radio-ulnar relationship

As the pin is driven up the medullary cavity of the radius, it meets certain resistance within the medullary cavity which gives to the surgeon a characteristic "feel" so that it is very easy to tell whether or not the pin has entered the medullary cavity rather than passing through the fracture line into the soft tissues

Checkup x ray pictures should be made while the head of the pin still emits through the stab wound, because if the pin has not been driven properly it is extremely difficult to grasp the head to remove the pin once it has been sunk beneath the skin covering

The best way to make checkup x ray pictures is to slip the cassette beneath the sterile drape being careful that the films are made

exactly in the antero-posterior and in the lateral planes

When it is certain that reconstruction is complete, and the pin is properly placed the head is sunk beneath the soft tissues using a small nail punch until the head grasps the cortex snugly. Care must be taken that it not be driven too deeply. If the head is driven completely into the bone substance there will be loss of fixation. On the other hand if the head is not driven home sufficiently it will protrude into the soft tissue and cause irritation on motion of the wrist joint. Usually the skin wound can be closed with one small stitch. From the appearance of the roentgenograms and from the feel of the wrist, it can be determined whether or not fixation is stable enough to omit plaster splints. If there is much swelling or soft tissue damage splints should be applied for

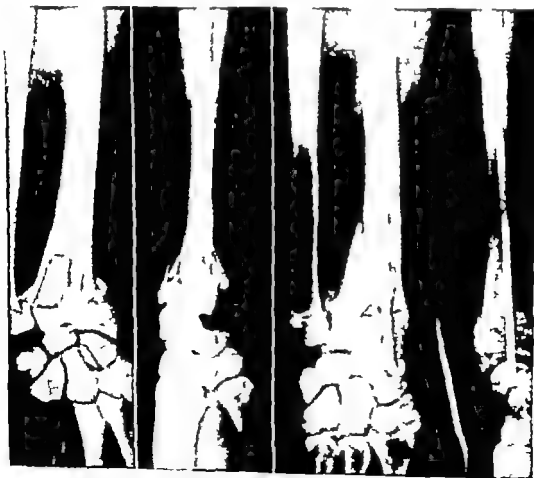


FIG. 5 Severely comminuted Colles reconstructed by medullary pin. Supplemental plaster splints were applied with elastic bandage. After one week the splints were removed periodically for exercise

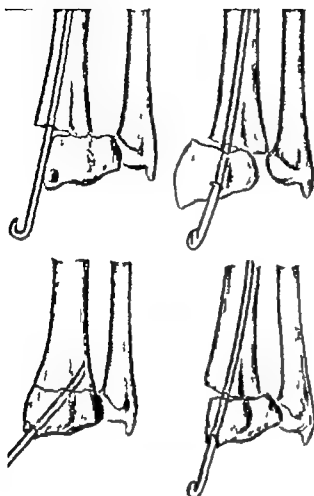


FIG. 6 Technical errors (A, Top left) Pin missed channel in distal fragment, result ulnar deviation of fragment with widening of the wrist. (B Top right) Point of entrance in distal fragment too far medial result radial deviation of distal fragment with poor proximation with loss of ulna relationship (C, Bottom left and right) Pin enters distal fragment at improper angle result angulation with disturbance of the radio-ulnar relationship Wrong angle of insertion can produce ulnar deviation (above) dorsal angulation or posterior angulation of distal fragment.

a few days for the comfort of the patient and to give him the needed confidence so that he will embark properly upon active motion.

STABILITY OF FIXATION

In most cases fixation is surprisingly stable. But stability can be influenced by many factors. The most important is the accuracy of the reduction and the placing of the pin. In the elderly the styloid end of

the radius can be only a shell and telescoping of the bone can occur. In this age group Colles fracture is rarely simple, and there are usually many small fragments.

CHART 1 REVIEW OF 50 CONSECUTIVE CASES COLLES FRACTURE

Average age	56 years
Oldest	93 years
Youngest	24 years
Women	42 cases
Men	8 (5 violent injuries)
Fixation completely stable	43
Fixation unstable	7
No external splints	46
External splints applied	4

ANATOMIC RESULTS

Gross deformity did not occur in this series. Shortening of the radial styloid and recurrence of the silver fork deformity were conspicuously absent. In no case was there radial deviation of the wrist. Recurrent luxation of the ulna persisted in one case.

In 4 cases there was some permanent deformity suggestive of a reverse Colles fracture—exaggerated radial arch with increased angle of inclination of the articular surface of the radius with dorsal convexity of the radius and with dorsal prominence of the styloid end of the ulna.

Silver fork deformity would have recurred in 4 cases had not supplemental splints been used. It has been encountered in cases not included in this series.

DYNAMIC FACTORS

A discussion of dynamic factors in medullary pinning is beyond the scope of this paper but it is necessary to call attention here to some of the factors involved.

For stability of fixation it is necessary for the pin to secure a good purchase upon both fragments. This is easily accomplished in the shaft of the radius because it is a mildly tortuous bone and when a medullary pin is driven into the shaft of the radius it secures bone contact at 3 points which gives firm fixation within this fragment.

For stability of the short distal fragment

there must be accomplished a successful opposition of forces. There is an intrinsic dynamic force at work in the wrist which produces and maintains the deformity of radial deviation. No matter what method is chosen to maintain the reduction of the fractured wrist, this must be accomplished by 3-point pressure whether applied externally or internally. With medullary pinning this 3-point pressure is applied within the substance of the bone.

As the pin enters the medullary cavity of the shaft of the bone the sled runner point is deflected toward the line of the long axis of the shaft. The shaft of the pin rocks on a fulcrum like a seesaw so that the head or proximal end of the pin is forced medially as the pin is driven in such fashion that the dynamic force exerted by the pin resists the intrinsic dynamic force of the extremity.

An additional opposing force is provided by the styloid end of the ulna, which resists the dynamic force of the pin in such fashion that if reduction is accomplished perfectly the radio-ulnar relationship is firmly re-established, and with the styloid end of the ulna snugly pressed into its notch in the radius this joint becomes stable despite the fact that the triangular ligament has been damaged.

Furthermore if one observes closely the contour of the distal end of the radius it will be noted that there is a natural bone gutter into which the proximal portion of the pin fits; this prevents anterior or posterior displacement of this fragment upon the pin. When the hooked head of the pin is driven down properly to grasp the cortex, the fixation becomes even more secure.

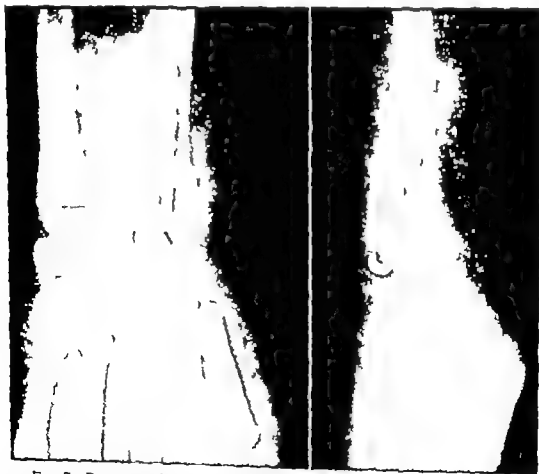


FIG 7 Reverse Colles type deformity caused by overcorrection of fracture with improper introduction of pin. Pin introduced too far medially on the dorsum. Had it been placed nearer the styloid process, this angulation could have been prevented. Note abnormal dorsal prominence of styloid end of the ulna.



FIG. 8 (Top) Anteroposterior view of Colles' fracture. Pin did not get good purchase on distal fragment. (Middle) Radius is sagging, with recurrence of silver fork deformity and loss of normal angle of inclination at the wrist. (Bottom) Deformity prevented by application of molded splints (film retouched)

Attention must be called to the fact that a sizeable fragment of the styloid process is essential for pin fixation, but it is a surprising and pleasant experience to see comminuted fractures in this region compressed into relatively normal relationship by the application of these dynamic forces.

TECHNICAL ERRORS AND COMPLICATIONS

In the entire series of 89 cases there have been no infections and no signs of embolism. In one instance the extensor tendon of the thumb was caught by the pin and resulted in some permanent loss of extension of the thumb. In this case the pin was introduced from a wrong position on the dorsum of the wrist rather than from the styloid.

On two occasions the head of the pin was not driven completely home and some

mechanical irritation occurred about the head of the pin making it necessary to remove the pin after the passage of several months. In several cases overcorrection of the distal fragment in ulna deviation with abnormal lengthening of the radial styloid was produced temporarily because the pin missed its passage in the distal fragment, passing through the soft tissues lateral to the bone. If this mishap is recognized from the roentgenogram at the time of operation, permanent deformity may be prevented.

Especially in young adults in whom the cancellous bone is firm and dense, the pin must pass through the distal fragment as nearly in correct alignment with the long axis of the bone as possible. The passage of the pin at an improper angle can result in angulation of this fragment to produce dorsal angulation anterior angulation or exaggerated ulnar deviation.

ray machine in no way supplants careful clinical examination. It is a fairly safe rule that if an examining surgeon will go over an injured extremity lightly digitally asking the patient for the point of greatest tenderness and having that point roentgenographed he will seldom be in error.

Most rapid swellings about the wrist are due to torn blood vessels, which, as a rule occur on the dorsum. They are treated best by a splint and a compression bandage (pref-

erably ACE elastic) elevation cold and local injection of 500 units of hyaluronidase. Continued active use of the fingers is important.

It is a mistake to feel that these hematomas are collected in a fascial compartment and can be relieved by aspiration. The hemorrhage is generally a diffuse intercellular one. About once a year one sees an overly ambitious intern who feels that these dorsal wrist hematomas could have a clot evacuated by



FIG. 3 A and B. (A Top) In recumbency the acromioclavicular joint appears to be normal. (B Bottom) Instability of the acromioclavicular joint is demonstrated by traction with the patient standing.

leave the pin in place permanently, removing it only if it should cause trouble.

DISCUSSION

In this series of 89 cases a critical study of 50 consecutive cases only was made because of the time limitation for this publication. In evaluating the results of this series several factors must be borne in mind. First, the series includes all cases which presented themselves regardless of the age group or the severity of the injury. Many of the fractures were either crush type or comminuted. Most of the cases are in the older age group, the average age being 56. All were private cases and the average hospital stay was 2 days.

Many of the patients thought that they were well and did not return for follow up treatment as requested.

Several cases included in the series were pinned from 2 to 3 weeks after the injury following unsuccessful attempts to hold the bones in proper position by external fixation. It might be mentioned here that it has been found possible to pin these cases satisfactorily after a passage of 3 weeks. One case was pinned 5 weeks after injury with but slight tendency to recurrence of the original deformity.

SUMMARY

- 1 A technic for closed medullary pinning of Colles' fracture is presented.
- 2 Technical errors and complications are discussed.
- 3 Conclusions are drawn from 89 cases.
- 4 Fifty consecutive cases are reviewed critically.
- 5 Physiotherapy was not used.
- 6 Gross deformity did not occur. Fixation was stable in 43 cases; splints were used in 4 cases.
- 7 Full function was regained by approximately 84 per cent of the patients within 4 weeks after surgery. Five patients showed some residual functional disability for 3 months or longer.

REFERENCES

- 1 Rush, L. V., and Rush, H. L. Longitudinal pin fixation in Colles' fracture of the wrist, *South Surgeon* 15: 679, 1949.
- Evolution of medullary fixation of fractures by the longitudinal pin. *Am. J. Surg.* 78: 324, 1949.
- Medullary pin fixation of fractures near joints, *Mississippi Doctor* 27: 260, 1949.
- Rush, L. V. Dynamic factors in medullary pinning of fractures, *Am Surgeon* 17: 803, 1951.
- Atlas of Rush Pin Technique (Book Section) *Mississippi Doctor* 31: 1 (July) 1953 and monthly installments to July 1954.

A Comparative Clinical Study of Autogenous and Frozen Homogenous Bone in Grafting Procedures

ERNEST A. BRAV, Colonel, MC U S A *

The refrigerated bone bank is now an established implement in the management of orthopedic problems. Its evolution can be traced to the bone grafting researches of John Hunter Ollier Barth, Lexer Axhausen and Macewen and to the important studies in tissue preservation by Alexis Carrel. Following the early clinical studies of bone grafting by Albee, Gallie and Robertson, Hey Groves and many others, the concept of bone storage for subsequent use was stimulated by the reports on chemically treated *os purum* by Orell and the refrigerated bone employed by Alberto Inclan. This principle was developed and popularized by Bush and his associates, and their refrigeration technic has received widespread adoption.

The advantages of such an available supply of bone grafting material requires no elaboration. However there is considerable difference of opinion regarding the relative value of such frozen, preserved, homogenous bone compared with fresh autogenous grafting material. There is conflicting experimental evidence concerning the physiologic activity and the rate of assimilation of these two types of bone. Some studies^{1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79,80,81,82,83,84,85,86,87,88,89,90,91,92,93,94,95,96,97,98,99,100} indicate that autogenous and frozen

homogenous grafts behave in identical fashion when transplanted, and that the end result is the same. Other observations^{1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79,80,81,82,83,84,85,86,87,88,89,90,91,92,93,94,95,96,97,98,99,100} on the microscopic tissue changes in grafted areas and on the physicochemical responses of the two types of bone grafts suggest that the osteogenetic quality of autogenous bone is superior and its incorporation is more rapid and complete.

Any conclusions as to the relative usefulness of these two types of grafting material must await the clinical results of a large series of parallel and well-controlled bone grafting procedures. As part of such a comparative series we instituted a plan for alternate grafting operations with fresh autogenous and frozen homogenous bone. This proved to be impractical because there was insufficient bank bone available for use in alternate cases and there were some patients on whom the use of bank bone was a necessity. Consequently we have succeeded in obtaining comparable series of grafting procedures but not alternating series. Seventy-five operations of similar types have been performed in each of the 2 groups during the past 3 years, and the minimal period of postoperative observation in the study has been 1 year.

Our bone bank has been patterned after the method of Bush and Garber. An ordinary deep-freeze unit is kept in our central

Letterman Army Hospital, San Francisco, Cal.
Presently Chief Orthopedic Service, Walter Reed
Army Hospital Washington, D. C.

service and is maintained at minus 30° C. No extra personnel are required for its operation, and aside from the original cost the method is entirely practical and inexpensive. Precautions as to continuation of the desired temperature are fulfilled by the sound of the motor, by a red light which indicates that the current is operating and by a warning bell which signifies an ineffective temperature. Our source of bone has been entirely from operative procedures and has been principally cancellous. All our open reductions of fractures are accompanied by iliac bone grafts and usually we are able to remove a small amount of extra bone for the bank. We have not yet solved the problem of the use of autopsy bone which is the obvious source of accumulation. Donors are checked for syphilis, malignancy and active tuberculosis. They are questioned concerning a history of malaria or hepatitis and are eliminated if any of these factors is positive. Bone specimens removed at operation are placed originally in a separate compartment of the freezer until the wounds of the donor are checked for infection. If prompt healing occurs, the bone is moved to another compartment and is made available for use. When needed, the bone is removed from the bank just prior to surgery and usually it is well thawed when ready for use. If it has not thawed it is immersed briefly in warm saline. Once bone has been removed from the bank it is not replaced. Any excess bone is discarded.

We have not stored massive cortical fragments, and when the grafts are used they are either ground up in the bone mill or transferred as small chips or slabs consisting principally of cancellous bone. We have avoided the use of preserved bone in operations for the bridging of large shaft defects. These restrictions may account in part for our successful results.

Originally we made an attempt to check the sterility of preserved bone by sending a specimen to the laboratory for culture at the time of operative removal. Fifteen positive cultures were reported in a short period of time and this bank bone was discarded. As confirmatory evidence we compared these positive laboratory reports with the healing of the donor wounds and with additional cultures made from the corresponding bank bone material before it was discarded. The gross inconsistency of this comparison (Table 1) suggested that in many of the cases there was little justification for discarding the bone and that other criteria for disposal should be adopted. In cases where there is infection of the donor site the discarding of the preserved bone seems to be entirely proper. However 12 of the 15 which were discarded were from donors whose wounds healed promptly. There were 8 in which the cultures of the specimens sent to the laboratory at the time of removal were compared with cultures reported from the corresponding preserved bone in the bank. In 4 of these the organisms reported by the labora-

TABLE 1 ANALYSIS OF INFECTED BONE SPECIMENS IN 17 CASES

DISPOSAL	NUMBER	DONOR SITE	BONE SPECIMEN CULTURE	BONE BANK CULTURE	RECIPIENT SITE
Discarded	1	Infected			
Discarded	1	Infected	Staph		
Discarded	1	Infected	Sterile	Staph	
Discarded	3	Healed	Staph.		
Discarded	4	Healed	Similar organisms		
Discarded	4	Healed	Different organisms		
Discarded	1	Healed	Diphtheroids		
Used inadvertently	1	Healed	Staph.		Healed
Used inadvertently	1	Infected			Staph.

tory as being in the specimens were entirely different from those found in the bulk of the refrigerated bone. In 2 patients, bone which was considered as infected was used inadvertently in grafting procedures. The specimen which the laboratory reported as being contaminated was removed from a donor site which healed without incident, producing no infection in the recipient. The other was transferred from a patient whose donor site became infected. The wound of the recipient was contaminated with the same organism that was cultured from the donor site.

In addition, a study was made of bank bone preparations which were considered as sterile because they were removed under aseptic precautions from donors whose wounds healed without infection. In 10 consecutive grafting procedures a specimen of such bone was sent to the laboratory at the time the bone was used. The results of these cultures were correlated with the healing of the respective recipient wounds. Three positive cultures were reported, and in each of these the wound of the recipient healed without infection. It is possible that our routine postoperative use of antibiotics in these

grafting procedures may have a bearing on the apparent contradiction of the laboratory evidence by the prompt wound healing. However, such therapy did not prevent operative wound infection in a patient whose grafted bank bone specimen was reported as sterile (Table 2). We have concluded that the conflicting laboratory and clinical evidence indicates that the discarding of bone removed under aseptic precautions is not warranted if the donor wound heals uneventfully. Any attempt to make repeated cultures from the bone bank in order to ascertain persistence of sterility entails an added possibility of contamination and seems to us to be unjustified.

STATISTICAL DATA

During the 4-year period of operation of our bone bank there have been 196 bone specimens stored. Twenty-seven were discarded: 15 for suspicion of infection and 12 because of interruption in refrigeration. Results have not yet been evaluated in 17 operations in which bank bone was employed and there were 13 specimens of bone remaining in the bank at the time of preparation of this report.

TABLE 2 BACTERIOLOGIC STUDY OF BANK BONE USED AT OPERATION

NO	DONOR SITE	TYPE OF DONOR BONE	AGE OF DONOR BONE (DAYS)	BACTERIOLOGIC REPORT OF DONOR BONE	RECIPIENT WOUND
1	Healed	Fibula	104	No growth	Healed
2	Healed	Ilium	135	No growth	Healed
3	Healed	Ilium	70	No growth	Healed
4	Healed	Ilium	33	Non-hemolytic Staph. aureus Alcaligenes sp	Healed
5	Healed	Ilium	52	No growth	Healed
6	Healed	Ilium	55	Non-hemolytic Streptococcus	Healed
7	Healed	Ilium	39	No growth	Hemolytic
8	Healed	Rib	148	Non-hemolytic Staph. aureus Non-hemolytic Streptococcus	Staph. aureus Healed
9	Healed	Ilium	77	No growth	Healed
10	Healed	Ilium	46	No growth	Healed

There were 139 donor specimens used in the 75 grafting procedures which are included in this study. One hundred and five specimens were removed from the ilium, 17 from the ribs, 4 from the outer end of the clavicle, 3 from the lower ulna and the remainder from cancellous areas such as the patella, the acromion process of the scapula, the astragalus and the humeral condyles.

The number of donor specimens used at operation varied between 1 and 10 with an average of 1.8. Since operations in which multiple specimens were employed presented an added risk of contamination we analyzed our infected cases in the light of this possibility. Of the 7 patients with wound infections, 3 were grafted from multiple donors while the other 4 had single donor grafts. The bank bone which was used in these 75 operative procedures had been stored for periods varying between 2 and 224 days with an average of 48.0 days. It is conceivable that the age of the preserved bone might have some influence on the success of the grafting procedure so that the 5 failures in the bank bone group were investigated from this point of view. All of the failures were found to have occurred following the grafting of bone which had been preserved for a relatively short period, in all cases less than 3 months whereas many of the successful procedures had been grafted with bone which was stored for much longer periods.

Type of Operation. In the 75 patients who had autogenous bone grafts, 46 were fresh fractures which were treated by open reduction, 3 presented delayed bone union, and 12 were patients with frank nonunion of fractures. There were 14 patients who had grafting procedures associated with major arthrodeses. In the bank bone group there were 29 patients with recent fractures, 10 with delayed union and 9 with nonunion of fractures. Seven spinal fusions were performed with bank bone. There were 14 patients who had major arthrodeses and 6 patients who presented bone cysts or tumors which were treated by curettage and packing with bank bone chips.

Analysis of Operative Results. A comparison of the end results in the 2 groups of patients reveals that in each there was prompt healing of bone at the grafted area in 63 patients. Delayed union was noted in 6 patients with autogenous grafts and in 7 patients with bank bone grafts. There were 6 (8.0%) operative failures with the use of autogenous bone and 5 (6.7%) with bank bone. It may be of some significance that 3 of the 6 failures with autogenous bone were associated with frank wound infection whereas none of the failures with bank bone presented such a complication. Since there were 6 infections in the autogenous group and 7 in the bank bone group there is the suggestion that the latter material may be somewhat more effective in the presence of infection, whereas in the clean cases the autogenous bone may be superior. Of course, the series is too small to offer data of statistical significance.

An analysis was made of the unsuccessful operations in the 2 groups in an attempt to obtain some indication as to the cause of failure and the results are shown in Table 3. Concerning the type of operative procedure in the 2 groups there was little difference in the incidence of failure. It is interesting to note that the percentage of failure

TABLE 3 COMPARATIVE BONE GRAFT SERIES.
ANALYSIS OF OPERATIVE FAILURES

	AUTOGENOUS GRAFTS	BANK BONE GRAFTS
TOTAL FAILURES	6 (8.0%)	5 (6.7%)
Type of operation		
Recent fracture	2 (4.1%)	2 (5.1%)
Old nonunion	3 (25.0%)	2 (22.2%)
Spinal fusion		1 (14.3%)
Arthrodesis	1 (7.1%)	0
Possible causes		
Wound infection	2	
Poor skin	1	
Subsequent		
Injury	1	
Unknown	2	5
Inadequate surgery?		
Quality of graft?		

with the use of bank bone in the patients with recent fracture is nearly as high as the failure rate for the entire group. This might be used in answer to any criticism of the grafting of recent fractures as a determination of the effectiveness of preserved homogenous bone. It is noteworthy that of the 6 failures in the autogenous group, 4 might be attributed to infection or to injury subsequent to operation. In none of the bank bone cases was there any apparent explanation for failure. Acknowledging the possibility of many variable elements one of which is the adequacy of the surgical procedure itself the implication remains that the quality of the grafting material may be the unknown factor in the operative failures of the bank bone group.

Analysis of Infections. There were wound infections in 6 (80%) operative procedures in the autogenous group and in 7 (93%) of the bank bone cases. Any

patient who presented a postoperative complication in wound healing however mild and due to whatever cause was included as

TABLE 4 COMPARATIVE BONE GRAFT SERIES
—ANALYSIS OF INFECTIONS

	AUTOGENOUS GRAFT	BANK BONE GRAFT
TOTAL INFECTIONS	6 (80%)	7 (93%)
Type of operation		
Recent fracture	4	1
Old nonunion	2	3
Bone cyst		1
Arthrodesis	0	2
Possible causes		
Compound injuries (27)	4	(19) 3
Poor skin	1	
Direct infection		1
Unknown	1	3
Technical errors?		
Graft contamination?		



FIG 1 (A Left) Anteroposterior and lateral roentgenograms showing nonunion of gunshot fracture of lower end of femur (B Right) Films 7 months after intramedullary fixation with supplemental wire loops and the use of cancellous bank-bone grafts.

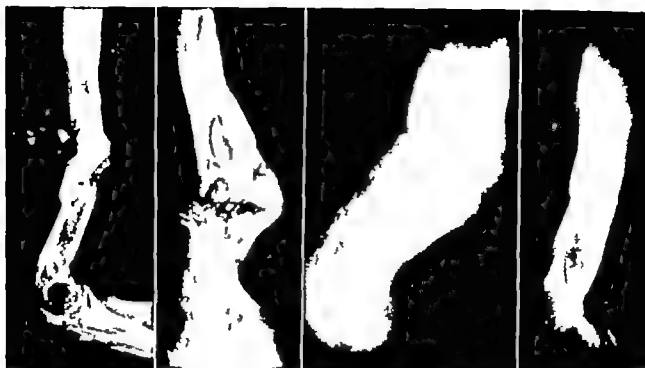


FIG. 2. (A *Left*) Anteroposterior and lateral roentgenograms of precarious bone union following gunshot fracture of lower end of humerus. (B *Right*) Enlarged views of same area 2 years after the application of reinforcing cancellous bank-bone grafts. The grafts are well incorporated and bone architecture has been restored. (Letterman Army Hospital Photographic Laboratory San Francisco)

a wound infection. Most infections were mild, and only 3 were associated with operative failure all of these being in the autogenous group. Table 4 represents an analysis of the infected cases in an attempt to study the cause of the infections. It is felt that the type of operation was of no importance in the incidence of infection. The slight difference in the 2 groups, especially in the case of recent fractures represents simply the somewhat different proportion of patients treated by the two methods. The incidence of infection was also the same in the 2 groups as related to the total number of

patients who formerly had open wounds and therefore presented a somewhat greater risk of subsequent postoperative wound infection.

One patient in the autogenous group had a skin condition which we feel was a predisposing factor to the infection. In 1 patient of the bank bone series, a graft was used from a donor site which became infected postoperatively. This bone should have been discarded. Of the 6 patients with infection in the autogenous group there remained only 1 in which no apparent reason for the infection existed and for which operative technical error must be held respon-

TABLE 5 RESULTS OF PRESERVED HOMOGENOUS BONE GRAFTING

	WEAVER	SICARD AND BINET	WILSON	REYNOLDS AND OLIVER	PRESENT SERIES	
Year reported	1949	1950	1951	1951	1953	
Number of operations	49	205	253	129	75	75
Type of preservation	Frozen	Frozen	Frozen	Merthiolate	Frozen	Fresh autogenous
Infection rate (per cent)	8.1	3.0	4.5	8.5	9.3	8.0
Operative failure (per cent)	17.3	14.0	15.0	30.2	6.7	8.0

sible. Of the 7 infections in the bank bone group there were 4 in which technical errors might be held accountable. One of these was definitely due to a contaminated bank-bone graft. While the other 3 may have no relation to the sterility of the preserved bone-graft material, we cannot entirely eliminate such a possibility whereas there is no comparable situation in the autogenous group.

DISCUSSION

The reported results of preserved homogenous bone grafting obtained by other observers in large groups of patients are represented in Table 5. Our failure rate compares favorably with that obtained elsewhere and we attribute our successful results to the use of principally cancellous bone applied in chips or slabs and to the avoidance of bank bone in the bridging of large shaft defects. Our experience with the progress of healing in the successful bank bone cases has been entirely gratifying. We have no reason to believe that for practical purposes a successful bone grafting result is any less rapid with bank bone than with autogenous bone and we are unable to distinguish between the two types of graft on the roentgenogram. Assimilation of the bank bone and restoration of local bony architecture apparently occur in a manner as satisfactory as with autogenous bone (Figs. 1 and 2).

In spite of these favorable impressions, a careful analysis of our failures and infections suggests that when factors such as wound infection and trauma are eliminated, there seems to be a somewhat better prospect for healing with the use of autogenous bone. Where the probable cause of failure was unknown, the incidence with bank bone was three times as great as with autogenous bone (Table 3). It would seem also that there is a greater risk of operative wound contamination with the use of preserved homogenous bone. However the latter seems to be more resistant to wound infection and appears to be preferable in the grafting of previously infected bone cavities or wherever the reactivation of an old wound infection is to be

feared. It must be emphasized that these impressions are based on a series of patients which is too small to be of statistical importance. Definite conclusions must await the results of a more extensive analysis of similar parallel groups of bone grafting procedures.

SUMMARY

Experience with parallel series of fresh autogenous and frozen homogenous bone grafting procedures has resulted in the following impressions:

- 1 The operation of a refrigerated bone bank is simple, practical and inexpensive.

- 2 Although the incidence of postoperative infection is roughly similar in the two groups, the possibility of wound contamination appears to be greater with preserved bone.

- 3 The percentage of operative failures is about the same in the 2 groups but when possible factors such as infection and trauma are excluded there is a considerably higher incidence of success with autogenous than with frozen homogenous grafts.

- 4 In the successful cases the progress of bone healing is apparently the same in the two groups and they are indistinguishable on the roentgenogram.

- 5 In the presence of infection the outlook for bone healing appears to be better with the use of frozen homogenous bone. This may compensate for the increased risk of contamination when such bone is employed.

- 6 When bank bone is utilized it is advisable to rely principally on cancellous bone which is divided into small slabs and chips. It is not recommended for the bridging of large shaft defects.

- 7 Fresh autogenous bone of normal quality is always preferable whenever available for grafting procedures with the possible exception of those in which the reactivation of an old wound infection is feared.

- 8 As supplemental bone for extensive grafting procedures or where satisfactory autogenous bone is not available, refrigerated homogenous bone can be employed with excellent prospects for success.

REFERENCES

- 1 Abbott, L. C., Schottstaedt E. R., Saunders, J. H. de C. M., and Bost, F. C. The evaluation of cortical and cancellous bone as grafting material an experimental study *J Bone & Joint Surg* 29 381-414 1947
- 2 Albee F. H. Transplantation of a portion of the tibia into the spine for Pott's disease *J.A.M.A.* 57 885-886 1911
- 3 Axhausen G. Histologische Untersuchungen über Knochentransplantation am Menschen *Deutsche Ztschr. Chir* 91 338-428 1908
- 4 Barth, A. Ueber Histologische Befunde nach Knochentransplantationen *Arch. klin. chir* 46 409-417 1893
- 5 Böhrer J. and Rupp G. Weitere Erfahrungen mit der Knochenbank, *Arch. orthop u. Unfall-Chir* 45 164-168 1952
- 6 Brooks, B. and Hudson W. A. Studies in bone transplantation. An experimental study of the comparative success of autogenous and homogenous transplants of bone in dogs, *Arch. Surg* 1 284-309 1920
- 7 Bush L. F. The use of homogenous bone grafts, *J Bone & Joint Surg.* 29-620-628 1947
- 8 Bush L. F. and Garber C. Z. The bone bank, *J.A.M.A.* 137 588-594 1948
- 9 Campbell, C. Brower T. and McFadden, D. G. Experimental study of the fate of bone grafts, in *Surgical Forum of Clinical Congress of Am. Coll. Surgeons*, pp 425 428 Philadelphia, Saunders, 1953
- 10 Carrel, A. The preservation of tissue and its application in surgery *J.A.M.A.* 59 523 526, 1912
- 11 Coley H. L. and Higinbotham N. L. Use of bank bone in the treatment of central lesions of bone *Am J Surg* 78 587 590 1949
- 12 De Bruyn, P. P. H. Bone formation by fresh and frozen transplants of bone bone marrow and periosteum, *Abstr. Anat. Rec* 99 641 1947
- 13 Galile W. E. and Robertson, D. E. The transplantation of bone, *J.A.M.A.* 70 1134-1140, 1918
- 14 Ghorimley R. K. and Stuck W. G. Experimental bone transplantation with special reference to the effect of decalcification, *Arch Surg* 28 742 770 1934
- 15 Gordon, H. and Welsh B. A bone bank Procurement, preparation and storage of accessions, *Am. J. Clin. Path.* 21 114-117 1951
- 16 Groves, E. W. Hey An experimental study of the operative treatment of fractures, *Brit. J Surg.* 1 438-501 1914
- 17 Harmon P. H. Experience with the use of a bone bank in 131 cases, *Permanent Found. M. Bull.* 8 97 106 1950
- 18 Herbert, J. J. and Paillet, J. Les greffes osseuses conservées par réfrigération. Résultats et indications, *Mém. Acad. chir* 76, 372 376 1950
- 19 Hunter John. *Collected Works*, London, J. F. Palmer 1817
- 20 Hutchinson, J. The fate of experimental bone grafts and homografts, *Brit. J. Surg.* 39 552 561 1952
- 21 Hyatt, G. W. Fundamentals in the use and preservation of homogenous bone, *U. S. Armed Forces M. J.* 1 841-852, 1950.
- 22 Inclan A. The use of preserved bone graft in orthopedic surgery *J Bone & Joint Surg.* 24, 81 96 1942
- 23 Keith, W. S. Small bone grafts, *J Bone & Joint Surg* 16 314-330 1934
- 24 Klehn, C. L., Friedell H. Benson, J., Berg, M. and Glover D. M. A study of the viability of autogenous frozen bone grafts by means of radioactive phosphorus, *Tr. Am. S. A.* 68 107 111 1950
- 25 Kreuz, F. P. Hyatt, G. W., Turner T. C., and Bassett, A. L. The preservation and clinical use of freeze-dried bone *J Bone & Joint Surg.* 33A 863-872 1951
- 26 Le Cocq J. F. Le Cocq E. A., and Anderson, K. J. Preliminary Report on the Use of Bone Bank Bone *Surg. Gynec. & Obst.* 91 277 280, 1950
- 27 Levander G. Über die Knochenregenerationische Fähigkeit des Periosts, *Acta chir scandinav* 83 1 26 1939
- 28 Lexer E. Free transplantation *Ann. Surg.* 60 166-194 1914
- 29 Macewen, W. The osteogenic factors in the development and repair of bone, *Ann. Surg.* 6 289 306 1887
- 30 ——— The Growth of Bone Observations on Osteogenesis An Experimental Inquiry into the Development and Reproduction of Diaphyseal Bone, Glasgow James Maclellan & Sons, 1912.
- 31 McWilliams, C. A. Bone grafting, *Internat. Abstr. Surg.* 22, 1 50 1916
- 32 Marrangoni, A. G. The fate of frozen homogenous bone transplants, *Am. J. Surg.* 82 378 380 1951
- 33 Odell, R. T., Mueller C. B., and Key J. A. Effect on bone grafts of radio-active isotopes of phosphorus, *J Bone & Joint Surg* 33A 324-331 1951
- 34 Okelberry A. M. Experiences in the use of frozen bone, *West. J. Surg* 59-385 389 1951



FIG. 4 Ethyl chloride spray for neck sprains

incision and drainage. Such ambitious youngsters choose the accident room for their supposed surgery, make an ill-advised incision, fail to find any large blood clot, encounter diffuse oozing and, before the days of penicillin, would end up with a serious tendon infection. So learn by their sad experience do not incise sprains in the hope of evacuating a large clot.

The troublesome "shoulder sprain" is a subluxation or dislocation of the acromioclavicular joint. It is a common injury in football, ice hockey and other similar body contact games. The usual mechanism is the pinning of the point of the shoulders between two forces, viz. a football lineman, who is pinned between two opposing linemen, or between an opponent and the ground. The neck yoke and the shoulder caps of football harness are designed specifically to prevent this type of injury yet they do not always accomplish it. Dislocations of the acromioclavicular joint are troublesome to the physician as many such injuries reduce themselves spontaneously in recumbency. As a result roentgenograms made in this position are reported as negative. If the roentgenogram is made with the patient standing (Figs 2 & 3) with weights in each hand the dislocation may be demonstrated. The treatment for luxations is immobilization for 3 to 4 weeks.

The treatment for complete dislocation is surgical.

Traumatic wry neck is commonly called a "neck sprain." Following a sudden twist or wrenching of the neck, a painful stiffness of the cervical spine may occur causing the head to assume the position of torticollis (Fig 4). Roentgenograms are negative. A clinical thermometer is a more valuable aid in diagnosis, as is the use of a tongue-depressor blade in ruling out reflex forms of torticollis such as early tuberculosis of the spine, or cervical lymph nodes, cervical adenitis, deep-seated inflammation of the neck, the pharynx, or the tonsils, as well as certain other types of tumors in this same region. True torticollis is painless.

When the wry neck is clearly post-traumatic, the mechanism of injury becomes of vital importance in determining the prognosis. If the force causing the trauma is a sudden turn or wrench of the head, i.e. without external force such as a handball player might incur by turning his head backward too suddenly in order to follow the ball, the condition clears spontaneously in about 10 days.

When the force causing the trauma is external, as in whiplash injuries of the neck, commonly caused by automobile collisions, particularly those in which one car is rammed from the rear, the prognosis is more serious because of the commonly encountered complications of cervical radiculitis and cerebral concussion. Discussion of whiplash injuries is beyond the scope of this chapter.³

While minor neck sprains clear up spontaneously in approximately 10 days, they are acutely painful, causing the patient to consult his doctor in order to obtain relief. Frequently dramatic relief is obtained by the application of ethyl chloride spray for a few minutes¹ (Fig 4).

Sprains of a more extensive type require cervical traction, either intermittently (Fig. 5 A) or continuously (Fig. 5 B) as indicated. An adjunct in therapy is local support by the use of a Thomas collar. As a general rule, minor sprains are fairly comfortable during the day even without support, but rest

- 35 Ollier L. *Traité expérimental et clinique de la régénération des os et de la production artificielle du tissu osseux*, Paris Masson, 1867
- 36 Orell S. Surgical bone grafting with "os purum" "os novum" and boiled bone *J Bone & Joint Surg* 19:873-885 1937
- 37 ——— Principles and experiences at the implantation of os purum, os novum and bone granulate *Acta orthop belg* 18 162 174 1952.
- 38 Orr H W. The history of bone transplantation in general and orthopedic surgery *Am. J Surg* 43 547 553 1939
- 39 Palmer L. Surgical treatment of defects of the long bones, *Acta chir scandinav* 193 381-400 1952
- 40 Reynolds F C and Oliver D R. Experimental evaluation of homogenous bone grafts *J Bone & Joint Surg* 32A 283 297 1950
- 41 Reynolds, F C., Oliver D R. and Ramsey R. Clinical evaluation of the merthiolate bone bank and homogenous bone grafts, *J Bone & Joint Surg* 33A 873-883 1951
- 42 Rountree C. R. Use of bone bank bone in bone surgery *South Surgeon* 16 966-980 1950
- 43 Sicard, A., and Binet, J P. La conservation des transplants osseux et leur emploi en chirurgie *Presse méd* 58 433-434 1950
- 44 Stuck, W G., and Dandridge, W S. Uses of refrigerated bone on a large fracture service *Am J Surg* 80:696-702, 1950
- 45 Weaver J B. Experiences in the use of homogenous (bone bank) bone, *J Bone & Joint Surg* 31A 778-792, 1949
- 46 Wilson P D. Experiences with a bone bank, *Ann Surg* 126 932 946 1947
- 47 ——— Experience with the use of refrigerated homogenous bone *J Bone & Joint Surg* 33B 301 315 1951
- 48 Zimbron, M A Velasco. Banque d'os. Greffe osseuse homologue. Etude de 128 interventions chirurgicales effectuées, *Mém Acad chir* 76 619-623 1950

The Use of Tubadil (Repository Injection of Tubocurarine) in Acute Back Strain

COMMANDER JOHN S THIEMEYER JR., (MC) USN AND
LIEUTENANT E F REED, JR (MC) USNR

Few problems encountered by the orthopedic surgeon are more refractory to treatment than back strain accompanied by severe pain and muscle spasm. Usually the patient presents a history of stooping and lifting an object and feels a "giving way" of the back or a sudden sharp and severe pain in the back. He then usually notices marked limitation of motion and pain frequently he has to go to bed to seek relief from the pain. Most of the patients described in this report had this mode of onset and were treated by the usual regimen of local heat, massage and rest on a firm bed, without significant relief in complaints. It was believed that treatment should be aimed more specifically at relaxation of the associated muscle spasm as rapidly as possible. With this in mind the Orthopedic Department of the U S Naval Hospital Portsmouth Va. tried Tubadil (repository injection of tubocurarine) which has a direct and rapid muscle relaxing effect by its tubocurarine action.

The use of curare dates back to the sixteenth century. It has been used in physiologic experimentation for nearly a hundred years. But it was not until about 15 years ago that preparations of curare, sufficiently purified for clinical use, became available.

d Tubocurarine from the plant *Chondrodendron tomentosum* was introduced into modern medicine by Gill. Through research by Burman,¹ Bennett,² Cullen³ and many

others it was found that the drug could be used for the alleviation of muscle spasm. Bennett reviewed the clinical investigations on curare in organic neurologic disorders in 1941 and noted that curare produced a marked relaxation of rigidity and abolition of involuntary movements in spastic paralysis, athetoid and dystonic states, parkinsonism, heredity chorea, spasmodic torticollis, status epilepticus and tetanus. Its action in aqueous solution was transient and fleeting. At a later date Fuller⁴ reported on the use of a repository injection of tubocurarine in painful muscle spasm due to traumatic injuries. The action of tubocurarine in the repository menstruum used by Fuller was apparent in 45 minutes, as indicated by relaxation of the spasm.

Tubadil repository tubocurarine, used in this study is manufactured by Endo Products Inc., Richmond Hill, N. Y. It contains a crystalline *d* tubocurarine chloride of high purity incorporated in a slow absorption base, capable of delivering a clinically effective dose of curare at a constant rate below the level which produces adverse side effects. Tubadil contains 25 mg. of *d* tubocurarine per 1 cc. The recommended dosage is individualized on a basis of body weight, 1.25 cc. per 70 kilograms or 154 pounds. The muscle relaxing effect is apparent in 45 minutes and continues for from 12 to 24 hours or more.

Certain measures should be followed to obtain the maximum usefulness of the drug. Anyone who uses it should be familiar with the signs and symptoms of overcurarization and of measures to combat this immediately. In the event of annoying side reactions, such as heaviness of the eyelids, diplopia or marked general muscle relaxation, the injection of $\frac{1}{2}$ to 1 cc of neostigmine is effective. The contraindications to the use of curare-containing drugs are respiratory deficiencies, pulmonic disorders and renal dysfunctions. It must never be used in patients even suspected of having myasthenia gravis. The dose must be measured accurately before administration and given intramuscularly in the upper outer quadrant of the buttocks. The total dosage is given at one

site. The patient is advised to refrain from excessive muscular activity for several hours after the administration of the drug. External heat must not be applied to the body, as this would increase the absorption rate and the site of injection must not be massaged for the same reason.

REPORT OF CASES ILLUSTRATIVE OF THE ABOVE THERAPY

Case 1 R.L.S., White Male, Aged 42. This man was admitted February 18, 1953, because of back pain and was treated conservatively. He had become freely ambulatory about the ward, doing cleaning of the decks and light work in the galley. On March 18, 1953, he suddenly developed a wry neck, and any motion of the neck caused severe pain. There was a coincidental flare up of his low back pain. He would

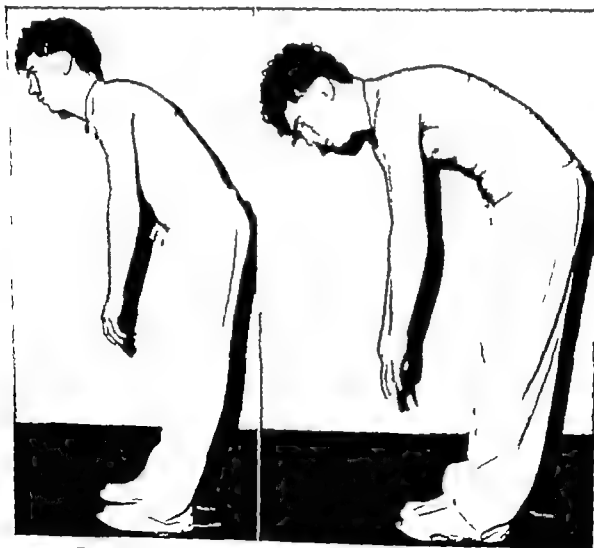


FIG. 1 (Left) Forward bending limited prior to receiving Tubadil.
FIG. 2 (Right) Increased forward bend one hour after receiving Tubadil.
(Official photograph, U S Navy)

not move from his bed, and in the bed he lay with his entire spine fixed.

At 11 05 A. M. 1 cc of Tubadil was injected into the muscle of the right buttock. Within 1 hour he was able to move his head about with little pain, and in 1½ hours he could move his head throughout a full range of motion without discomfort. The pain in his low back had decreased to a dull ache and he was able to move about the bed and the ward. At 1 00 P. M. he stated that he noticed difficulty in focusing his eyes properly. This diplopia persisted for about 3 hours following the injection. There was no recurrence of neck pain and stiffness, but the pain reappeared in his low back about 36 hours after his injection. Four days later the pain still was not as severe as prior to Tubadil and it subsided over a period of a week, and he returned to duty.

Case 2 H.F.S. White Male, Aged 37
This Navy Chief was admitted with a recurrent pain in the low back for the past 2 years. Diathermy and massage sometimes relieved the pain. Four days prior to admission he stooped to pick up a razor blade from the deck and was unable to straighten up. He had received the usual heat, massage and bed rest without relief. On examination he walked with a marked list to his left, with splinting of all muscles of the low back. He had difficulty in sitting or arising from a chair. He had to have assistance in dressing. There was muscle spasm of the lumbar muscles, more marked on the left. Straight leg raising to 45° caused pain in the back. There was no radiation of pain and no relation to expulsive efforts. Roentgenograms were negative.

He was given 0.75 cc of Tubadil in the left gluteal region. Half an hour later he stated that he felt more relaxed and on examination at one hour he was found to be taking a shower and had an almost full range of motion of his back without pain. He still had some list but walked about freely. He noticed a diplopia about 2 hours after injection but this lasted only about 2 hours. He received no other treatment, and one week later he was returned to full duty with no list, full back motion and no pain.

Case 3 R.D.A., White Male, Aged 24
This patient had pain in the low back of sudden onset which had lasted for about 1 week and did not respond to the usual regimen of heat, rest, etc. He had marked spasm of the para vertebral muscles, especially on the left, with almost poker stiffness of the spine on forward flexion. He could bend only to within 24 inches of the floor with the fingers extended and com-

plained of pain on any motion of his back. The Gaenslin test was positive for lumbosacral involvement.

At 12 05 P. M. he was given 0.75 cc. of Tubadil in the right buttock and put to bed. By 1 00 P. M. he stated that he had almost no pain, but he noticed some stiffness in turning over in the bed. At 2 00 P. M. he was able to bend with the fingertips to within 8 inches of the floor. Lateral bending, hyperextension and rotary movements were normal. All movements were pain free. He still stated that he felt some stiffness in the low back and examination revealed some limited motion of the lower thoracic spine. He had a slight drowsiness, but no diplopia or vertigo. This man was observed until 4 00 P. M. and allowed to return to his ship.

Case 4 R.T.H. White Male, Aged 20
This young man was admitted on March 22, 1953 with the diagnosis of "contusion of low back," having suddenly experienced backache when lifting a heavy object. He had marked muscle spasm and limitation of all motions of the back. He was given 0.75 cc of Tubadil in the right buttock and in one hour he stated that all the pain had disappeared except on extremes of motion. The spasm had entirely disappeared. He had a "full feeling" in his head, but no diplopia or vertigo. Photographs made before and after injections are presented (Figs. 1 and 2). He was returned to duty on March 27, 1953 with a full range of back motion and no pain.

Case 5 F.H.M., White Male, Aged 19
This man was a boxer from Little Creek Amphibious Base who felt sudden and sharp pain in his low back while boxing. Heat, massage and rest did not relieve the pain or the spasm. On examination he had marked spasm of the left sacrospinalis and could flex only to about 150° with no hyperextension of the back demonstrable. Lateral bending was to about 20° and there was no rotation of the spine. All motions were accompanied by considerable pain in the low back. He was given 0.75 cc. of Tubadil in the right buttock, and within 1 hour all muscle spasm was gone. He complained of difficulty in focusing his eyes on distant objects and of an extreme dizziness but was able to walk well with no weaving and no tremor. Three hours later he had full lateral bending, hyperextension and rotation of the spine and was able to bend forward to within 8 inches of the floor without discomfort. Diplopia lasted for about 2 hours after injection. He was returned to duty 4 hours later and boxed the next night.

Case 6. F.W., Aged 46 This man had a sudden onset of back pain following lifting and was treated by 3 days of bed rest with little improvement. He had marked limitation of all motions of the back with considerable bilateral erector spinae muscle spasm. He was given 0.75 cc. of Tubadil in the buttock at 2:00 P.M. and 2 hours later had almost full range of pain free motion of the back. He continued to have some discomfort on full flexion, and the finger tips lacked 6 inches of touching the floor. Twenty four hours later the patient asked to be returned to duty as he was completely asymptomatic.

Case 7. C.D.N., White Male, Aged 30 This patient noted recurrent low back pain of unknown origin for several months previously which became acute in the last 3 weeks. Roentgenograms revealed no evidence of pathology. Examination revealed considerable muscle spasm of the lumbar muscles with radiation of pain into the posterior thighs and limited lumbar motion in all planes. Tubadil, 1.25 cc. was given at 10:30 A.M. and by 2:00 P.M. he was relaxed and able to tie his own shoes for the first time in 3 weeks. He had little or no pain on leaving the hospital at 4:00 P.M.

Case 8. J.L.H., White Male, Aged 22. This patient had pain of the low back following shearing stress which occurred about 4 weeks previously and was not relieved by the usual conservative means. This occurred when he was picking up a heavy object, and at that time he felt a sudden, sharp pain of the low back. Roentgenograms of the lumbosacral spine were negative. Physical examination revealed considerable muscle spasm and marked limitation of all motions of the lumbar spine. One cc. of Tubadil was injected into the buttock and within 2 hours his lumbar spasm was gone. The following day he was freely ambulatory with only slight stiffness of the low back, but no pain, and on the fifth day after injection he returned to full duty. He had a transient diplopia for about 2 hours following injection of Tubadil.

Case 9. J.C.S., White Male, Aged 19 This patient fell from a ladder aboard ship a distance of about 4 feet and struck his buttocks. He developed pain in his low back which persisted for about 6 weeks and was not relieved by rest, local heat or the usual conservative means. Roentgenograms of the lumbosacral spine were negative. Physical examination revealed spasm of the lumbar muscles and marked limitation of motion of the lumbar spine especially in forward flexion. He was given 1 cc. of Tubadil

intramuscularly at 10:40 A.M. A diplopia developed within 1 hour and persisted for about 3 hours. In 2 hours the muscle spasm was relieved and the range of pain free motion was 75 per cent of normal. By the following morning he was ambulatory and pain free. He still could not touch his fingertips to the deck but could reach to within 4 inches. Six days later without repeat injection he returned to duty fit for same.

Case 10. R.D., White Male, Aged 25 This patient had acute pain and marked limitation of motion of the lumbar spine of 8 days duration following lifting a heavy object. This was not relieved by the usual conservative measures. Roentgenograms of the lumbosacral spine were normal. He presented spasm of the paravertebral muscles and only about 10% of motion of the low back. He was given 1 cc. of Tubadil and within 2 hours had 50 per cent of normal range of motion of the low back and very little pain. The following day the injection was repeated, and 3 hours later he was able to touch the deck with his fingertips. Two days later without further treatment, he went to full duty.

SUMMARY

Twenty cases of low back strain with pain and paravertebral muscle spasm but no bony abnormality demonstrable clinically or by x rays, nor with evidence of neurologic disease were treated by intramuscular repository injection of *d* tubocurarine. Only 2 were not improved, and these were thought to have had functional overlay. Two others were only partially relieved and the remaining 16 were greatly improved. Three had two injections and the remainder had only one injection. Usually hospitalization overnight was not required, but it is advised that if this treatment is used, the patient should be retained under observation for 4 hours or longer. In this series less than the recommended dosage has been used averaging 0.75 cc. for a 150-pound patient, as it was found that the desired results were obtained. Half of the patients treated noticed a transient diplopia beginning in about 30 minutes and lasting about 3 hours after injection and one complained of a marked vertigo that cleared in 2 hours. Other than these no untoward

effects were noted, nor was neostigmine required in any case.

It is the opinion of the authors that Tubadil has a definite place in the armamentarium of physicians treating acute back strain with muscle spasm and back pain.

REFERENCES

- 1 Burman M S Curare therapy for the release of muscle spasm J Bone & Joint Surg 20 754 1938
- 2 Bennett A. E. Clinical investigations with curare in organic neurologic disorders Am J M Sc. 202 102, 1941
- 3 Cullen S C. The use of curare for the improvement of abdominal muscle relaxation during inhalation anesthesia, Surgery 14 216 1943
- 4 Fuller J D Use of slowly absorbed suspension of *d*-tubocurarine in traumatic injury J.A.M.A. 143 789 1950
- 5 Hoback, W W Repository tubocurarine in trauma., J Tennessee M A. 45 16-18 1952

Fatigue Fracture of the Shaft of the Femur— Report of a Case*

HYMAN R OSHEROFF, COLONEL, MC, U S A , AND
THOMAS C DEVLIN, First Lieutenant, MC, U S A

Fatigue fractures are relatively common in Army personnel. Frequently they are seen involving the metatarsal bones. March fracture of the foot is a clinical entity and is diagnosed easily by the typical roentgen findings of a minute fracture line surrounded by exuberant callus. However, when bones other than the metatarsals are involved, the diagnosis is not reached so readily. At first the lesion usually is considered as a bone tumor. In 1942 Peterson† reported a case of fatigue fracture of the shaft of the femur. Prior to his publication only 1 such case had been reported; thus it would seem that this is an uncommon condition.

CASE REPORT

The patient, a 20-year-old white male private, was seen first by a dispensary doctor at this installation. He complained of pain and tenderness of the left thigh, localized at a point about 2 or 3 inches above the lateral femoral condyle. He gave no history of injury. He stated that he had had this pain almost constantly for 3 weeks. The patient had been undergoing basic training and had been subjected to long periods of marching and physical training. He could not remember any distinct trauma to the left leg. The

pain was described as being dull and aching in nature; it was made worse by physical activity but not completely relieved by bed rest. Occasionally it awakened the patient at night.

Physical examination was essentially negative with the exception of the left lower extremity. There was a definite hard swelling which appeared to be deeply situated just above the flare of the condyles of the left femur. This was very tender. There was a full range of motion in the hip and the knee. There was no evidence of ecchymosis or any other soft tissue involvement in the left lower extremity.

The patient was admitted to the hospital April 27, 1953, for surgical observation. Roentgenograms taken on admission revealed a small fracture line in the posterior cortex of the femur with elevation of the periosteum in this area and amorphous calcification extending into the soft tissues. Because of the periosteal reaction about the femoral shaft, it was felt that a malignancy of bone was a good possibility. Laboratory studies were done and the results were as follows: WBC 12,600; neutrophils 75; lymphocytes 17; monocytes 7; and eosinophils 1. Hemoglobin was 13.40 G; Calcium was 10.8; phosphorus, 3.6; alkaline phosphatase, 1.9. The urinalysis was negative.

In view of the negative laboratory findings and since additional roentgenograms of

U. S. Army Hospital, Camp Gordon, Ga.

* Peterson, L. T. March fracture of the femur. *J. Bone & Joint Surg.* 24:185, 1942.

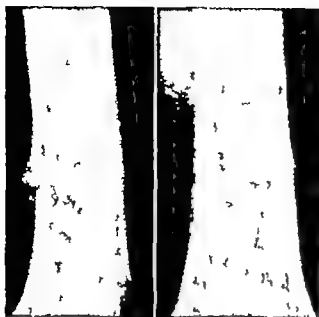


FIG 1 AP and lateral roentgenograms taken on admission April 27 1953 showing fracture of the posterior cortex, periosteal elevation and amorphous calcification

the femur showed the presence of a minute fracture line traversing the shaft of the femur it was decided to watch and wait. Therefore the patient was kept at bedrest. In a few days his pain gradually subsided



FIG 2 Roentgenograms taken May 11 1953 showing increase in callus formation and partial obliteration of the fracture line



FIG 3 Roentgenograms taken August 31 1953 showing complete healing of fracture

although the swelling persisted and this area remained tender. Roentgenograms were obtained again approximately 3 weeks later. These revealed obliteration of the previously mentioned fracture line. What was at first thought to be periosteal reaction was on these films thought to be normal callus formation. Eventually the patient was allowed ambulation and was able to get about well without any recurrence of his pain. Final roentgenograms were made about 4 months later; these revealed complete healing of the fracture. The patient was returned to limited duty July 10 1953 and as yet has had no recurrence of his symptoms.

SUMMARY

The above case is being reported because of its rarity and because of the tendency to mistake this type of pathology for a bone tumor. We seriously considered a biopsy of this bony mass when the patient was first seen, and it was only after serial roentgenograms had been taken that we arrived at a correct diagnosis and did not subject this man to a useless operative procedure.

Vascular Relations of the Ankle and Their Clinical Significance*

H. KELIKIAN, M D

The ankle is a weight bearing joint. It connects two unequal levers. The foot joins the leg at an angle and moves on it around a single axis. The axis of movement runs from side to side across the angle of union of the leg and the foot. It passes from a point slightly below the lowermost blunt border of the tibial malleolus to the tip of the fibular malleolus. As the lateral buttress of the ankle is placed posteriorly and fathoms the entire depth of the body of the talus and the tibial malleolus extends forward and descends only one third down the medial side of the astragalus, the axis of hinge motion inclines downward and backward from the inner to the outer border of the ankle. Around it the talus rocks up and down. Since no muscles connect with it, the talus can move only by the gear action of the contacting bones or by the tension of the attached ligaments. It is either pushed or pulled.

When the foot is at a right angle to the leg, the angle formed between the weight bearing axis of the tibia and the long axis of the talus is about 100°. From this position the talus moves 15° dorsally and 35° plantarward. Due to increments of gliding movement allowed in joints distal to the talus, the foot as a whole moves through a

greater arc 30° up toward the leg and about 50° away from it. Dorsiflexion is carried out with greater ease when the knee is flexed and the tension on the tendo achillis is removed.

In plantar flexion and dorsiflexion, the line at which the foot folds on the leg is a bias. Because of the obliquity of the axis of hinge action and the tapering of the lateral wall of the body of the talus from front to back, in plantar flexion the head of the talus twists down and medially; in dorsiflexion it turns up and laterally. The foot follows suit and accentuates this corkscrew action of the talus. When the sole of the foot is depressed toward the ground, the gastrocnemius pulls the os calcis hence the heel, into slight varus; the forefoot deviates medially and the toes point down and in. Conversely when the dorsum of the foot is lifted toward the front of the leg, the heel returns to its normal slightly valgus position, the forefoot is deflected laterally and the toes point up and outward. Thus plantar flexion is accompanied by inversion and adduction (supination) and dorsiflexion by eversion and abduction (pronation) of the foot. The plane of movement of the foot against the leg is not altogether antero-posterior; it is spiral.

ARTERIAL PATHWAYS

As in other joints the main arteries which cross the ankle are four:

*From Department of Bone and Joint Surgery, Northwestern University Medical School, Wesley Memorial and Cook County Hospitals, Chicago.

ward which the segments move and when there are two vessels the larger is located on the side toward which movement is more extensive. Since the ankle permits greater range of plantar flexion than dorsiflexion the larger artery is placed behind and the smaller artery in front of the joint. Moreover in plantar flexion the sole of the foot faces backward and turns medially the posterior tibial artery traverses along the posteromedial aspect of the ankle. In dorsiflexion the back of the foot inclines laterally toward the fibula the anterior tibial artery descends from the lateral aspect of the leg down and medially.

Great Arterial Loop. Higher in the leg the popliteal artery bifurcates into two terminal branches the anterior and the posterior tibial trunks. The anterior tibial artery passes forward and enters the front of the leg through an aperture in the proximal end of the interosseous membrane. Here the artery is flanked by the tibia and the fibula and establishes for itself a point of fixation. At the bend of the ankle the tendon of the long extensor of the big toe crosses over the artery from the fibular to the tibial side and enters a special compartment provided by the inferior extensor retinaculum under the shelter of this tendon the anterior tibial artery gives off two malleolar branches and passes over the back of the foot as the *dorsalis pedis*. Just distal to the ankle over the neck of the talus, the *dorsalis pedis* shoots out a large tarsal branch in the direction of the outer border of the foot and then continues toward the first interosseous space. At the apex of this space between the first and the second metatarsal bones the continuation of the anterior tibial artery—the *dorsalis pedis*—turns down toward the sole of the foot. It connects directly with the lateral plantar artery which is the larger terminal branch and hence the more direct continuation of the posterior tibial artery which in turn is the larger terminal branch or the more direct continuation of the popliteal artery.

The two main arteries of the leg and their

continuation in the foot thus complete a large loop which is analogous to the circle of Willis within the cranium in that the anastomosis is through a large channel and not solely through capillaries. In general the plane of the arterial loop cuts across the skeletal framework of the leg and the foot and more or less parallels the twisted plane in which plantar flexion and dorsiflexion are carried out. The posterior limb of the great arterial loop—consisting of the tibialis posterior and the lateral plantar arteries—is placed on the side toward which movement is more extensive, it gives off a greater number of segmental branches in the leg and supplies the skin on the back of the ankle adequately. The anterior limb is smaller in caliber and closer to bone with no muscles intervening both at its inception and its termination it is flanked by bones, and at the bend of the ankle the extensor hallucis longus tendon crosses it from the lateral to the medial side. Thus the anterior limb is bound down and gives off fewer segmental branches, which—because they are placed deeply behind gliding tendons—do not reach the skin in profusion. The integument in front of the ankle, especially over the tibial malleolus, is not richly supplied with nutrient vessels.

Accessory Arterial Loop. The peroneal artery—an offshoot of the posterior tibial artery—passes down the leg and splits into two terminal branches. The smaller perforating peroneal artery penetrates the lower portion of the interosseous membrane and descends in front of the tibiofibular syndesmosis the direct continuation of the peroneal artery gives off several twigs which pass through and around the posterior talofibular ligament and connect with the perforating artery in front. The two terminal branches of the peroneal artery thus complete a circuit around the outer malleolus of the ankle. This constitutes the small or the accessory arterial loop of the ankle.

Lateral Tarsal Rete. As in dorsiflexion, the back of the foot inclines toward the fibular malleolus and the tissues immediately below that process and to the outside of the



FIG 5 (A *Inset*) Intermittent cervical traction. (B *Below*) Continuous cervical traction.

less nights may be avoided by the use of a Turkish towel folded 4 times longitudinally wrapped twice around the neck, and pinned with a safety pin to keep it in place

The low back will be discussed briefly The back situation is a complicated one and supposedly always will be confused Consider the case of the working man who develops a catch in his back. By the next morning he is in quite severe pain and is pulled or bent over to one side He states that he has sprained his back What should he do?

The first suggestion is that he consult his physician and that the physician instead of quickly prescribing adhesive tape heat treatment, aspirin, codeine or some other drug, should make a careful diagnosis

In most instances diagnosis will show the most common cause of such sprained backs to be fatigue which produces "muscle



FIG 6 Mechanism of typical American football knee injury

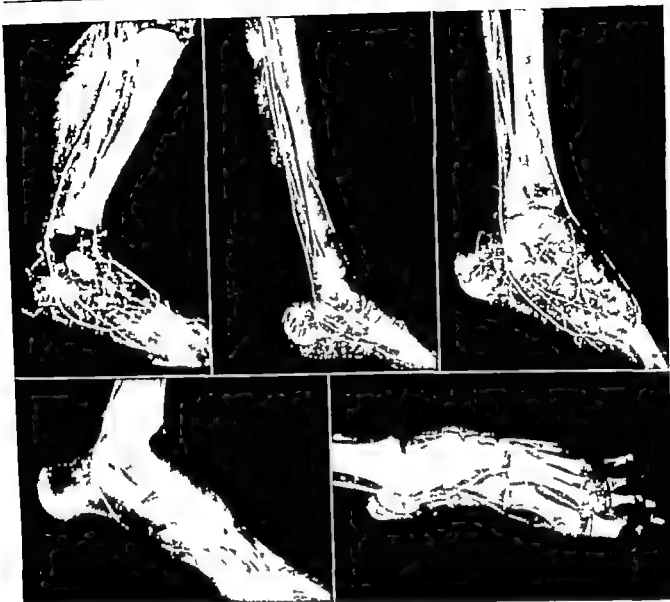


FIG. 1 Arterial pathways—arteriograms (A, *Top left*) The arterial loop in a new born. Note the relative size of the arterial trunks. (B *Top center*) The arterial loop in an adult (*Top right*) Distal portion of the arterial loop. Sizable tarsal twigs emanate from the dorsalis pedis and run laterally toward the outer neck of the talus. (D *Bottom left*) Terminal portion of the arterial loop showing a direct communication between the dorsalis pedis and the lateral plantar artery. (E, *Bottom, right*) Arteriogram in a case with congenital absence of the fibula. Note the missing anterior limb of the great arterial loop.

neck of the talus are comparatively lax a richer web of blood vessels is to be found here than in the corresponding area in front of the tibial malleolus, where movement is limited and the skin is snug. The anastomotic network is formed by the two terminal branches of the peroneal artery by the lateral malleolar twig of the anterior tibial artery by a large offshoot from the tarsal branch of the dorsalis pedis and by small recurrent filaments from the lateral plantar artery.

Tibial Malleolar Rete. The medial malleolar branches of the anterior and the posterior tibial arteries—one coming from in front and the other from behind—anastomose with one another over the base of the inner malleolus, establishing a small vascular side-sling around the bone. The anastomotic lacework lies behind the tendons, and its capillaries penetrate the bone just above the base of the tibial malleolus.

The arterial pathways described admit of

some variation. In two cases of congenital absence of the fibula the anterior tibial artery was completely missing, the *dorsalis pedis* was represented by a small thread coming down from the peroneal artery and it did not connect directly with the lateral plantar artery.

SUMMARY

The two main arteries of the leg and their continuation in the foot complete a large loop which is analogous to the circle of Willis in that anastomosis is through a sizable channel and not by way of capillaries alone. The posterior limb of the great arterial loop



FIG. 2 Arterial pathways. Roentgenograms from arteriosclerotics. (A, *Top left*) Lateral view showing tortuous anterior and posterior limbs of the great arterial loop at the level of the ankle. (B, *Top right*) This roentgenogram shows a large sclerotic artery between the first and the second metatarsal bones. This vessel connects the *dorsalis pedis*—the continuation of the anterior tibial—with the lateral plantar which is the continuation of the posterior tibial artery. (C, *Bottom left*) Lateral view roentgenogram showing the proximity of the *dorsalis pedis* to the neck of the talus. (D, *Bottom right*) The same after astraglectomy for pyogenic arthritis of the ankle. Note the collapse of the tortuous artery into the cradle of the extracted talus. Later this patient was subjected to tibiotarsal fusion, and the surgeon inadvertently cut the herniated segment of the artery. The foot survived by virtue of patent posterior tibial artery.

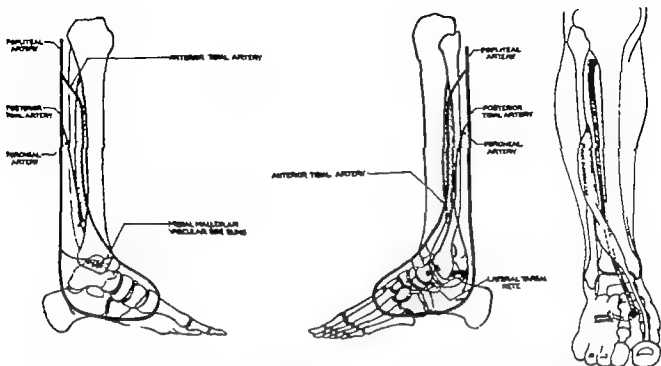


FIG 3 Arterial pathways. Drawings illustrating the great arterial loop and its accessions. (A, *Left*) The arterial loop viewed from the medial side (B *Center*) The arterial loop from the lateral aspect. (C, *Right*) The anterior limb of the loop in relation to bones and to the long extensor tendon of the great toe

is padded away from bone and gives more numerous segmental branches the anterior limb runs in the skeletal plane and gives off fewer side twigs. A lesser arterial loop is formed around the distal tibiofibular syndesmosis by the terminal branches of the peroneal artery. At the level of the ankle over and around the malleoli, one finds 2 anastomotic side-slings. A richer lacework is seen outside the neck of the talus where the tissues are lax. In 2 cases of congenital absence of the fibula the anterior tibial artery was missing (Figs 1 2 and 3)

RETURN CIRCULATION

The veins and the lymphatics which cross the ankle joint pursue a less definite pattern. The deep veins accompany the arteries as *venae comites* and therefore lie within the confines of the deep fascia; the superficial veins tend to avoid the bony knuckles, the malleoli. The smaller short saphenous vein which is a vestigial remnant of the postaxial or sciatic trunk of the embryonic limb bud curls up behind the fibular malleolus in com-

pany with the terminal branches of the sural nerve. The long saphenous vein—the original preaxial vein—is larger and courses up the medial side of the ankle in front of the tibial malleolus; it is accompanied by the terminal sensory branch of the femoral nerve.

The direction of venous and lymphatic flow in the foot is from the deep to the superficial system in the leg; the direction is reversed. The veins and the lymphatics of the lower limb empty themselves against gravity with the aid of the transmitted pulse from the arteries, the squeezing action of the contracting muscles, the massage of the gliding tendons and the movements of the joints. As no muscles except a few fleshy fibers of the peroneus tertius cross the ankle joint, the greatest aid to venous and lymphatic flow is eliminated and return circulation must depend upon the remaining factors. At the level of the ankle the superficial veins and lymphatics benefit mainly from the movements of the joint proximally around the calf; the impulse of muscular contraction as transmitted by the crural fascia also helps.

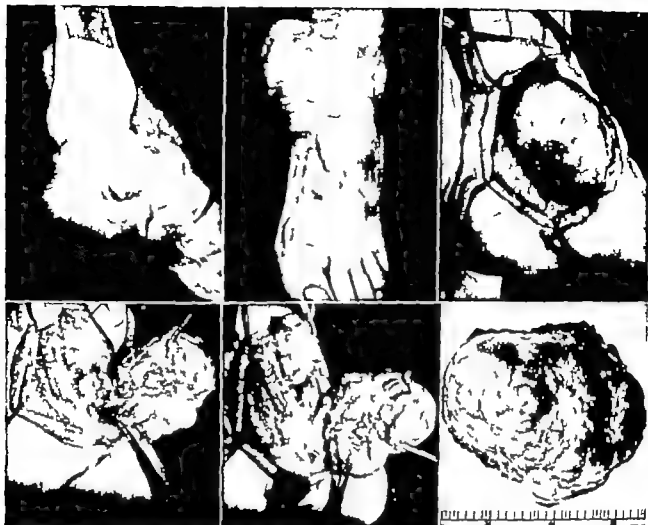


FIG 4 Return circulation. The ankle is dependent. It constitutes a common site of stasis and associated phenomena, such as skin and fascial thickening, ulcers and congestive osteitis; absorption from around the ankle is extremely sluggish. (A, *Top left*) Lateral view roentgenogram of a man who was shot by a soft lead bullet 21 years earlier. Apparently the lead was soluble which in a joint like the knee, where the absorption is quick, might have given rise to systemic symptoms. This man had no blood changes, no bleeding gums or peripheral neuritis. He did not even have any local discomfort. The tumor was removed for cosmetic reasons. Note that the dissolved or precipitated lead is held in suspension and is walled off. (B *Top center*) Frontal photograph of the tumor. (C *Top right*) Tumor partly exposed. (D *Bottom left*) Tumor dissected from the neck of the talus and the capsule of the ankle. Lower forceps points to the as yet undissolved lead. (E, *Bottom center*) The lead bullet has been removed. Note the hole within the neck of the talus and the ankle joint. The ankle was washed copiously with saline. (F *Bottom right*) The multilocular cyst which held the precipitated lead in suspension.

Should the deep fascia thicken, as after multiple surgical operations and per fascial fibrous deposits caused by inflammations of the veins and the lymphatics the muscles fail to transmit their squeezing action through the stiffened fascia, and stasis ensues.

After it leaves the front of the tibial malleolus, the long saphenous vein passes upward over the distal tibia and receives no

benefit from the transmitted impulse of the muscular contraction. This so-called silent area over the distal tibia just above the inner malleolus constitutes the most common site of stasis ulcers.

The deep lymphatic vessels run in close relation to the arteries as do the deep veins the superficial lymphatics accompany the saphenae. There are no lymph nodes around

the ankle. Absorption from the ankle is extremely sluggish as compared with the knee (Fig. 4)

CLINICAL CORRELATION

Avascular Necrosis in Dislocations and Fractures of the Talus. Information about the blood supply of the bones of the ankle—the talus included—is gained by consideration of the following (1) the growth pattern of the bones, (2) the extent of their articular coating (3) the grooves and the fulcra that they provide for tendons to glide over (4) the attachments they give and the tenseness and the laxity of the anchored ligaments (5) the proximity of the bones or portions of them to richer or poorer vascular networks and the density or the looseness of the intervening tissues (6) the size the number and the allocation of nutrient foraminae scattered over their nonarticular surfaces and finally (7) the behavior of the detached or displaced piece of bone in fractures and dislocations

1 **THE GROWTH PATTERN** The talus is derived from one primary center of ossification. Occasionally about the age of 7 or 8 a secondary center appears in the tail of the bone and is destined to become the lateral or trigonal tubercle. At times this center fails to effect a bony union with the main body then it is called os trigonum. The osseous core within the primary center appears at the seventh month of intra uterine life. Around it from the innermost aspect of the cartilaginous shell newer bone is deposited, layer by layer in a concentric fashion until the age of 20 when all but the articular cartilage surrounding the bone is consumed. The ossification center first appears in what is destined to become the neck of the talus. As bone must have blood vessels, the neck of the talus captures its provision of vascular supply early. At birth the body of the talus is mainly cartilaginous bone and blood vessels grow into it from the neck backward. As growth proceeds, the ankle bone changes in shape and inclination. In children the talus slants down and medi-

ally to a greater extent. In some African tribes the neck of the talus definitely tilts up and bears an articular facet on its superior aspect, which, on squatting, comes into contact with a cartilage-coated surface in front of the distal tibia. However as a rule the neck of the talus touches no other bone and bears no articular coating. Medially, both above and below the neck is narrowed by the encroaching articular surfaces of the body and the head laterally where a richer arterial network is to be found, the neck of the talus is spacious.

2 **THE EXTENT OF THE ARTICULAR COATING** Hyalin cartilage constitutes a barrier to blood vessels and the neck of the talus is the only portion of the bone that is not insulated by it. The neck provides the main surface area for penetration of nutrient vessels into the talus. The globular head in front is capped completely with hyalin cartilage, and the massive body behind is almost surrounded with it.

3 **THE GROOVES AND THE FULCRA** The neck of the talus is constricted and its surfaces are sunken. They proffer no fulcrum or groove for tendons to glide over. blood vessels can reach the neck without any frictional interference by gliding tendons.

4 **THE ATTACHMENTS** No muscles, only ligaments, connect with the talus. Unlike muscles ligaments are not lush conveyers of vessels to bone. The deltoid ligament which gains the widest attachment to the body of the talus is under strains and its fibers are disposed axially thus it carries only small twigs to the talus. The posterior talofibular ligament joins with the trigonal tubercle of the talus and its fibers run transversely and are not under strain. It carries sizable vessels, most of which pass forward to the fibular malleolus but some of which turn posteriorly toward the tail of the talus. When the trigonal tubercle remains unfused and presents itself as os trigonum, a thin band of fibrocartilage intercedes between it and the main bone and blocks blood vessels from passing into the body. The anterior talofibular ligament, which connects with the

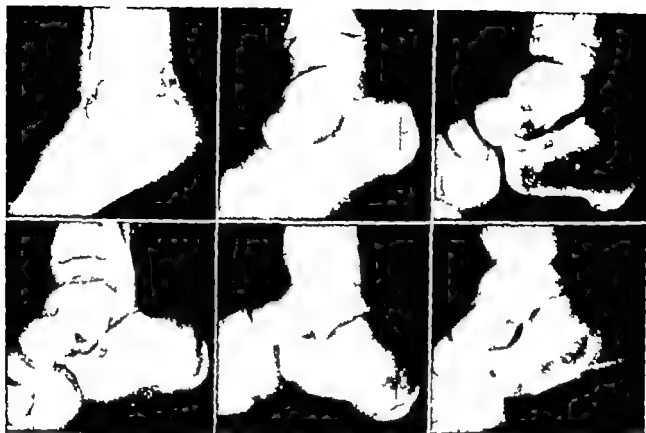


FIG 5 Ossification of the talus. (A *Top left*) Talus at 2 years of age. (B *Top center*) Talus at 4 (C, *Top right*) Talus at 6 (D *Bottom left*) Talus at 8 Note the appearance of the secondary center in the tail of the bone. (E, *Bottom center*) Talus at 11 The trigonal center is almost fused. (F *Bottom right*) Mature talus with growth arrest lines due to ingestion of phosphorized cod liver oil between the ages of 1 and 2. Note that considerable bone has piled up posteriorly on the osseous core delineated by the growth arrest lines in childhood.

outer neck of the talus also is disposed horizontally Except in extreme plantar flexion and inversion of the foot, it is not under strain. Moreover it is a weak band. The corresponding area on the medial aspect of the neck of the talus is hooked by the strong anterior fibers of the deltoid ligament, and a comparatively weak ligament and lax capsular structures connect with the outer neck of the talus they can convey sizable vessels to the bone

5 THE PROXIMITY OF VASCULAR NETWORKS AND THE LAXITY OF INTERVENING TISSUES In dorsiflexion the neck of the talus turns toward the fibular malleolus a richer network of blood vessels is to be found on the side of greater movement, next to the broader outer neck of the talus where the tissues are lax The lateral tarsal rete per haps contributes the greatest number of nu

trient vessels to the talus, since it is closer to the spacious outer neck of the bone

6 NUTRIENT FORAMINAE There is no one large nutrient artery which enters the talus there is no single sizable nutrient foramina There are many, some smaller than others. In freshly boiled specimens one sees numerous apertures around the neck as well as along the fringes of the body of the talus Only smaller holes are to be seen on the noncartilaginous portions of the body The neck is peppered with numerous foramina.

7 THE BEHAVIOR OF BROKEN BONES The talus presents certain peculiarities of structure and disposition which influence its liability and condition its reaction to injury The body of the talus sustains the entire weight transmitted by the tibia, but the talus as a whole is only partly supported by the

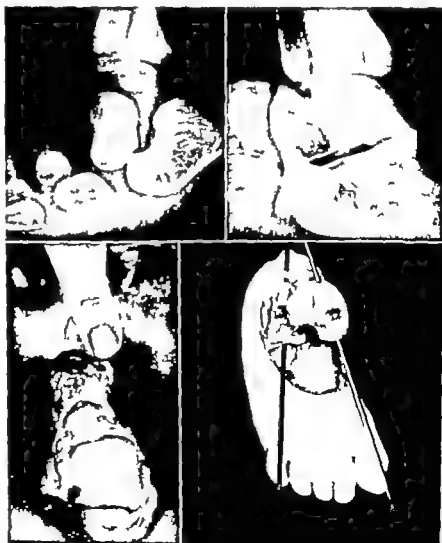
tarsal bones. Long accustomed to great pressure and cushioned around with cartilage, the body of the talus can withstand considerable compression. When forced, the body of the talus, and with it the entire bone slips out of the line of stress. It bears on the adjacent bones and breaks them. It tugs at the restraining ligaments and tears them. Surgeons of the past likened the talus to the slippery kernel inside a cherry when compressed it pushes its envelop aside and may tear its way through the integument. Dislocations of the talus are more frequent than fractures. Displacements of the astragalus

usually are accompanied by fractures of the surrounding bones. When the talus yields through its own substance, the line of fracture runs across the part of the bone which is not covered with cartilage namely, the neck.

Its lack of cartilaginous padding, its constricted shape and its precarious position between the hindfoot and the forefoot make the neck of the talus the most vulnerable portion of the bone. In fractures of the neck of the talus the effective blood supply of the body sometimes is cut off and the bone undergoes avascular necrosis. This is espe-

Fig. 6. Inclination and alignment during growth of the talus. The mature talus rides side-saddle on the os calcis. The disposition of the 2 tarsal bones can be simulated by crossing the long finger of the homolateral hand astride the index the tip of the third digit diverges medially its pulp projects without any support from below much like the under surfaces of the head of the talus, which rests not on bone but on ligament. In the coronal plane the talus turns medially while the os calcis points toward the outer

toes, and the long axes of the two make an angle of 40° in the newborn and 15° in the adult viewed from the side, the talus is definitely plantarflexed—more so in children than in adults and to a greater extent in fetals than in infants. Occasionally the fetal pattern will persist, and the talus remains plantarflexed. (A, Top left) Plantarflexed talus. Note the elongated neck, hence, the greater surface of area for penetration of blood vessels. (B Top right) Africoid talus. This type has a short neck which bears an articular facet superiorly and has precarious circulation. (C, Bottom left) Talocalcaneal alignment in the adult, indicated by crossing the third finger over the index of the homolateral hand. (D Bottom right) Talocalcaneal alignment in the newborn. Note the greater divergence of the long axes of the talus and the os calcis as indicated by Kirschner wires.



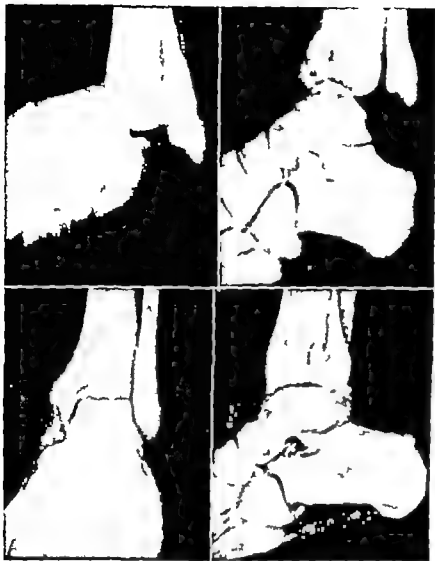


FIG 7 Dislocation of the talus away from the leg. (A, Top left) Anteroposterior view roentgenogram of the ankle of an obese woman who suffered a compound dislocation with the ankle joint gaping wide open laterally (B Top right) Lateral view (C Bottom left) Anteroposterior view — roentgenogram 18 months after open reduction and reconstruction of the deltoid ligament, using half of the tibialis posterior tendon for tenodesis. The patient recovered with no traumatic flatfoot. (D Bottom, right) Lateral view Talus shows no sign of avascular necrosis.

cially true if the body fragment is displaced as well. Since the body of the talus enters into the two main weight bearing joints—the ankle proper and the posterior talocalcaneal articulation—avascular necrosis and consequent crumbling of the bone and degenerative arthritis become real hazards. In fractures of the body of the talus when the plane of break cuts across two articular facets segregating a cartilage-coated piece of bone, avascular necrosis is even more imminent. In simple dislocations when the unbroken talus slips out of its wonted location and carries with it fractured pieces of the surrounding bones, it retains most of its capsular and ligamentous attachments, hence its blood supply and lives.

Transcervical fractures of the talus without dislocation may be treated conservatively

until such time if ever when avascular necrosis of the body becomes established, then ankle or tarsal fusion is performed. Present trend inclines toward triple arthrodesis to vascularize the dead talus. However arthritis of the ankle is more disabling than arthritis of the subtalar joint. Triple arthrodesis is resorted to when pain persists after a stable ankylosis of the ankle. In transcervical fractures with partial displacement of the body, it is best to reduce the dislocation by two-pin traction and to immobilize and elevate the parts. When the skin takes on a healthy appearance and x ray films denote a turn toward avascular necrosis, ankle fusion is resorted to. In arthrodesing the ankle, the foot is displaced backward so that the denuded distal bearing surface of the tibia rides astride the body and the neck, with



FIG. 8 Dislocation of the talus away from both the leg and the foot (A Left) Anteroposterior view showing counterclockwise rotation of the talus out of the ankle socket and medial displacement of the foot at the talocalcaneal and the talonavicular joints. (B Center) The same patient a year after reduction. (C, Right) Lateral view roentgenogram. Talus shows evidences of avascular necrosis.

FIG. 9 Fractures and fracture-dislocations of the talus in which avascular necrosis is almost certain. (A, Top left) Basal fracture of the neck of the talus involving a portion of the body. (B, Top right) Fracture of the body with the plane of break traversing across two articular facets and segregating a cartilage-coated piece of bone. (C Bottom left) Fracture-dislocation with partial extrusion of the body. (D Bottom right) Transcervical fracture with extrusion of squashed body out from under the pulverized tibial malleolus.

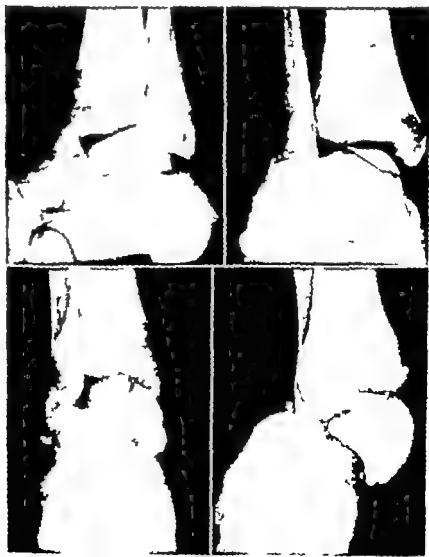




FIG 10 Laceration of the posterior limb of the great arterial loop. Paradoxical as it may seem, in some severe fracture-dislocations of the talus the posterior tibial artery—protected from bone by intervening muscles as it is—runs a greater risk of being lacerated than the anterior tibial which runs in the skeletal plane close to bone. This apparent discrepancy is explained by the fact that the posterior tibial artery passes under the taut flexor retinaculum in the posteromedial aspect of the ankle, where the tibio-fibular socket is defective. The broken body of the talus is extruded from behind the tibial malleolus and may injure the posterior tibial artery (A, *Left*) Transcervical fracture with extrusion of the body out under the flexor retinaculum (B *Center*) The same patient 6 weeks after open reduction (C, *Right*) Arteriogram of the same case. The posterior tibial artery is not visualized, but the foot is supplied adequately by the anterior limb of the great arterial loop.

the fracture line lying deep under the tibial vault. When the body fragment is completely extruded out of the ankle socket—especially when it lodges under the tense flexor retinaculum—the displaced bone is reduced by two-pin traction (one through the tibia, the other through the os calcis as shown in Fig 19 D) or extracted through an incision, and arthrodesis is postponed to a later date. In compound-fracture dislocations the detached, displaced body is removed, the wound is closed and tibiotarsal arthrodesis is performed later through viable skin. In fractures of the body when the plane of break traverses two articular surfaces, early arthrodesis of the ankle is advised (Figs 5-11).

Fractures of the Bones of the Ankle Socket. The criteria which were utilized in appraising the circulatory relations of the talus may be applied to the skeletal components of the ankle socket.

1 **THE GROWTH PATTERN.** The fibular malleolus is designed as a low guarding buttress to prevent the talus from slipping outward and backward. Its growth is unique.

The fibula is an exception to the general rule that the epiphysis away from which the nutrient artery runs ossifies first and unites last. The nutrient artery of the fibular shaft runs from the knee toward the ankle. Yet the ossification of the lower epiphysis of the fibula antedates the appearance of bone in the upper center by 4 years. This discrepancy is explained by the fact that the proximal center of the fibula anchors the powerful biceps tendon and is a traction epiphysis, much like the trochanters of the femur. As with all tension epiphyses, it appears late in life around the fifth year. The distal center of ossification of the fibula is designed to block the outward twist of the talus and is to be regarded as a pressure epiphysis as such it acquires its osseous nucleus early, toward the end of the first year of life. Since bone must have blood supply to thrive the osseous core of the distal epiphysis of the fibula captures its provision of blood vessels early at points where it is not covered by either conjugal or articular cartilage. The entire distal epiphysis of the fibula goes into the formation of the malleolus, and the



FIG. 7 Relationship of the menisci to the lateral hinges (A) internal semilunar fibrocartilage (B) external semilunar fibrocartilage



FIG. 8 Ethyl chloride spray to relax knee sprain

spasm" pulling the spine out of alignment. Anything to relax this muscle spasm makes the patient happy, including massage ("manipulative therapy") heat or injections of procaine or curare.

Such methods are superficial and represent symptomatic treatment only. Unless the underlying causes of fatigue are eliminated recurrences are inevitable. The question in the doctor's mind when examining a lame back should be "Why is this man tired?"

Knee injuries belong predominately to American football and as a result are encountered most frequently in adolescent boys. In Spain they kill bulls for entertainment; in America they tear up knee joints. In industry a falling stack of boxes or bales of paper frequently reproduce knee injuries similar to those of the football player (Fig. 6). The initial knee injury labeled a sprain is actually some form of tear of the internal lateral ligament and is rarely at first a dislocated cartilage. If this sprained knee is placed in a cast for 3 weeks healing occurs and the chance that a dislocated or torn cartilage will develop later is remote. However, neither the coach nor the boy wishes to miss the next Saturday game and neither the working man nor his boss wishes to run his safety record. The injured person bakes his knee, tapes it and attempts full activity in 3 or 4 days on a weak knee. A second injury occurs, and this time because of the intimate relation of the internal semilunar cartilage to the internal ligament, a cartilage tear results (Fig. 7) and the boy receives a serious knee injury. Summarizing: Initial knee injuries are generally torn internal lateral ligaments. Serious later injuries can be avoided by proper care.

In adolescent girls the knee sprain is due to their inept use of high-heeled shoes. The following example covers the usual pattern for this type of knee injury.

The mother calls up usually on Sunday morning stating that her daughter is an emergency case. The daughter is somewhere around 16 years of age and has developed a "locked" knee which is very painful. The patient hops along on her good leg to the



FIG. 11 Tear of the posterior tibial trunk and displacement of the anterior tibial trunk. The anterior limb of the great arterial loop is flanked proximally and distally by bones at the level of the foot it is overslung by the long extensor tendon of the great toe. This tendon and its fascial slings serve as a hedge along the medial aspect of the artery. They carry the artery laterally in outward dislocations of the foot the tibial malleolar branch of the anterior tibial artery may rip but the main trunk shifts unharmed toward the fibula. (A, *Top left*) Anteroposterior view of an old fracture-dislocation of the talus 3 months after closed reduction. (B, *Top right*) Arteriogram of the same case. The anterior limb of the great arterial loop which normally crosses the ankle about midway between the 2 malleoli, has been displaced laterally. Its fibular malleolar branch is visualized its tibial malleolar twig is not seen. The tibial malleolus is fractured there is considerable tibiofibular diastasis. (C, *Bottom left*) Lateral view. Note that the posterior limb of the great arterial loop is not visualized, and the tarsal twig emanating from the dorsalis pedis does not reach the neck of the talus. (D, *Bottom right*) Roentgenogram of the resected talus and the tibial malleolus. Compare the density of the body of the talus with that of the neck and the head. The inserted roentgenogram at the top of D is a film of the partly replaced tibial malleolus.

conjugal cartilage which links it proximally with the shaft runs flush with the horizontal line of contact between the bearing surfaces of the tibia and the talus the conjugal cartilage enters the ankle joint and is continuous with the articular facet which the malleolus presents to the talus medially. Thus the osseous nucleus is surrounded by cartilage above and over the upper portion of its medial surface, it must receive its nutrient supply from its lateral surface and from the fossa below the articular facet. In rare instances the lowermost pole of the fibular malleolus arises from a separate center of ossification and remains connected with the main malleolus by a semilunar band of fibrocartilage. In bipartite fibular malleolus the two centers receive their separate provision of blood supply.

The bearing plate of the distal extremity of the tibia arises from the same ossification center as the inner malleolus, and the conjugal cartilage which connects the common epiphysis with the shaft proximally is placed about 1 cm. above the transverse line of contact between the tibia and the talus. It lies on a higher level than the conjugal cartilage of the fibular malleolus and does not enter the ankle nor does it connect with the articular cartilage. First, a horizontal bar of bone is deposited in the core of the bearing plate of the distal tibia. This portion of the common growth center is rightly regarded as a pressure epiphysis since it is destined to bear the body weight. Like all pressure epiphyses, it appears early—during the first year of life—and captures its blood vessels which grow and extend with it.

The tibial malleolus is merely the continuous downward projection of the distal articular extremity of the tibia and it arises from the same epiphysis as the bearing plate. The malleolus gives anchorage to the deltoid ligament which also is attached to the talus inferiorly and laterally to the os calcis and the spring ligament below and to the navicular bone in front. The deltoid ligament restrains the tendency of the foot to twist outward around its long axis, and its sus-

pension point proximally is merely a traction beak drawn down from the inner edge of the distal growth center of the tibia. As a tensional outgrowth of an epiphysis and not as its pressure plate, the tibial malleolus acquires its osseous core late in life—about 6 years after the appearance of bone in the bearing plate or in the fibular malleolus. In girls at 7 and in boys a year later the transverse bar of bone within the distal epiphysis of the tibia begins to turn medially into the cartilaginous frame bone grows downward toward the tip of the malleolus, and vascular channels align themselves in that direction.

2 THE EXTENT OF THE ARTICULAR COATING The hyalin facet which the fibular malleolus presents to the talus occupies only a small area on the upper portion of its medial surface, it leaves an extensive fossa inferiorly where the stout posterior talofibular ligament lodges and where numerous blood vessels enter the bone.

Between the articular surface below and the conjugal cartilage above the bearing plate of the distal tibia is surrounded by a ring of nonhyalin surface where blood vessels can penetrate the bone.

The articular cartilage cements the entire talar surface of the tibial malleolus and is even reflected over its inferior blunt border thus limiting the area where blood vessels can penetrate the bone.

3 THE GROOVES AND THE FULCRA On its posterolateral aspect, the fibular malleolus offers a small groove for the peroneal tendons to glide. By far the greatest portion of the outer surface of the fibular malleolus is free of gliding grooves and is available to penetrating vessels.

Most of the tendons which pass in front of and behind the ankle bowstring across the ring of noncartilaginous surface surrounding the bearing plate of the distal tibia they offer no frictional interference to penetrating vessels.

On its posteromedial aspect the tibial malleolus bears a broad elongated groove for the tibialis posterior and the flexor digitorum

longus tendons, both of which hug the bone and glide over in long, extensive sweeps no sizable vessel can penetrate the bone in the presence of this fractional interference

4 THE ATTACHMENTS Of the three separate components of the fibular collateral ligament, only the middle, or calcaneofibular cord is under tension and has a small point of anchorage at the lowermost tip of the fibular malleolus. The other two—the anterior and the posterior talofibular ligaments—gain broader connections. Their fibers are disposed horizontally and are without undue tension in physiologic shifts at the ankle; they can convey sizable vessels to bone.

In front and behind the bearing plate of the distal end of the tibia gives diffuse attachments to the anterior and the posterior capsular ligaments, both of which are lax and can convey sizable vessels to the bone.

The tibial malleolus moors the powerful deltoid ligament which spreads fan-wise toward the tarsal bones but its stronger strands are disposed axially and therefore are under tension. Like all ligaments under stress the deltoid conveys only minute vessels to its more confined proximal point of anchorage along the inferior rim of the tibial malleolus.

5 THE PROXIMITY OF VASCULAR NETWORKS AND THE LAXITY OF INTERVENING TISSUES Both the accessory arterial loop and the lateral tarsal rete lie close to the outer ankle and the tissues in front and behind the fibular malleolus and the adjacent bearing plate of the tibia are comparatively lax.

The tissues around the tibial malleolus are snug. The closest network—the tibial malleolar rete—is comparatively small. The vascular side sling is placed on the skeleton itself and not on loose yielding tissues; it donates only minute filaments to bone.

6 NUTRIENT FORAMINAE In cleared specimens one sees numerous foraminae in the fossa below the small articular facet of the fibular malleolus. Both in front of and behind the ring surrounding the bearing plate of the distal tibia there are numerous aper-

tures, fewer and smaller foraminae are to be seen over the nonarticular portions of the tibial malleolus.

7 THE BEHAVIOR OF BROKEN BONES The fibular malleolus receives ample blood supply at almost all levels except at its very tip where the middle cord of the fibular collateral ligament—the calcaneofibular ligament—is attached. Like the deltoid on the medial side of the ankle, the calcaneofibular ligament is under tension, and its fibers run downward and backward in axial direction. The calcaneofibular ligament has a confined point of attachment at the tip of the malleolus and when under excessive stress, it pulls off a small piece of bone. In these so-called sprain fractures the plane of break falls below the level of penetration of the nutrient twigs into the infra-articular fossa. The detached ossicle of bone is comparatively avascular and fails to effect bony union with the main fragment proximally. With this one exception in almost all fractures of the fibular malleolus the line of break usually passes above the level of the penetration of the vessels into bone—above the fossa into which the posterior talofibular ligament is lodged. The distal fragment is well supplied, and there is no risk of avascular necrosis. Moreover the low-guarding fibular buttress is broken most often by compression, and the line of fracture may start at the base of the malleolus but runs up spirally or obliquely into the shaft; the malleolar fragment is large and there is a wide area of medullary contact. These fractures unite readily.

Only occasionally in comminuted fractures of the bearing plate of the tibia caused by axial compression, a cartilage-coated piece is cut off completely from the mother bone and dies. These fractures are treated best by fixed traction and cast and by prolonged nonweight bearing. Should painful arthritis intervene, arthrodesis of the ankle is advised.

In general then, vascular disturbance does not interfere with the healing of most common fractures of the fibular malleolus.

of the bearing plate of the distal tibia, both of which are pressure epiphyses. They capture their provision of nutrient supply early in life. blood vessels grow and extend with them and gain favorable distribution.

The tibial malleolus is a traction break. Bone and vascular channels grow into it late in life from the main bearing plate down toward the tip of the malleolus, articular cartilage insulates its entire talar surface down to its very tip and is reflected in part over its lower rim. The deltoid ligament which captures the remainder of the inferior border of the malleolus is under stress and conveys only minute vessels to bone on its posteromedial aspect, the tibial malleolus bears a broad sulcus for tendons to glide over which leaves only a small triangular area over the anteromedial surface of the malleolus where nutrient vessels can enter the bone. The small tibial malleolar rete from which these twigs arise is placed on the skeleton itself and not on loose, yielding tissues. The side-sling formed by the medial malleolar branches of the anterior and the posterior tibial arteries lies outside the periosteum with which the deep fascia of the leg blends in this region. Nutrient vessels pierce and transfix the periosteum and pass into the bone obliquely downward. Most of the vessels penetrate the bone in the limited area over its anteromedial surface about midway from the tip of the malleolus and the common conjugal cartilage. Since the latter is placed 1 cm. above the bearing surface and the malleolus itself descends that far the point of entrance of blood vessels into bone comes to life at or above the base of the malleolus—the site of avulsion fractures of the tibial malleolus.

Brief of length, blunt and rounded along its inferior border the tibial malleolus allows the foot to be pushed inward and often escapes compression. Inversion continuing the calcaneofibular ligament on the other side gives or the outer buttress of the ankle is pulled off before the tibial malleolus itself yields to the upward thrust of the talus. The line of break in these compression fractures

starts at the angle of union between the tibial malleolus and the inferior bearing surface of the tibia, it runs up and medially almost in a sagittal plane and cuts through where the shaft of the tibia begins to flare medially. The malleolar fragment is large, and the plane of fracture is above the level of the penetration of nutrient vessels into the bone. The broken fragment has ample supply of blood, and its fractured surface affords wide medullary contact. It lives and it unites.

However compression fractures of the tibial malleolus are rare. The tibial malleolus is broken more commonly by the pull of the deltoid ligament, which loosens the malleolus off its base. In these avulsion fractures the detached malleolar fragment is deprived of its medullary source of nourishment coming down from the bone above, and if the fracture line lies below the level of penetration of nutrient vessels at its base as it often does, the broken malleolus is cut off in addition from its surface source of blood supply. Often, avulsion fractures of the tibial malleolus are accompanied by outward dislocation of the foot. The long extensor of the big toe and its retinaculum carry the anterior tibial artery laterally away from the vascular side-sling formed by the medial malleolar branches of the anterior and the posterior tibial arteries. The malleolar branch of the anterior tibial artery runs the risk of being torn, the corresponding branch of the posterior tibial severed from its anastomotic mate is likely to retract. Thus the blood supply to the malleolus may be put out of function completely and the broken fragment of bone has to thrive on what little nourishment it obtains by way of the attachment of the deltoid ligament inferiorly. This source of supply is at best meager and precarious.

When recent avulsion fractures of the tibial malleolus are explored surgically and the broken surface of the malleolus is scraped by a curet it fails to bleed, while the surface of the bone above bleeds freely. Granulations cannot be expected to sprout

from the avascular malleolar side and vascular tissue growing down from the main bone often fails to engage the malleolar fragment and effect union unless reduction is accurate and fixation firm. In old, un-

ited fractures of the tibial malleolus caused by the pull of the deltoid ligament one often finds the malleolar surface white and pale and the fractured surface of the main bone sealed by an apron of fibrous tissue.

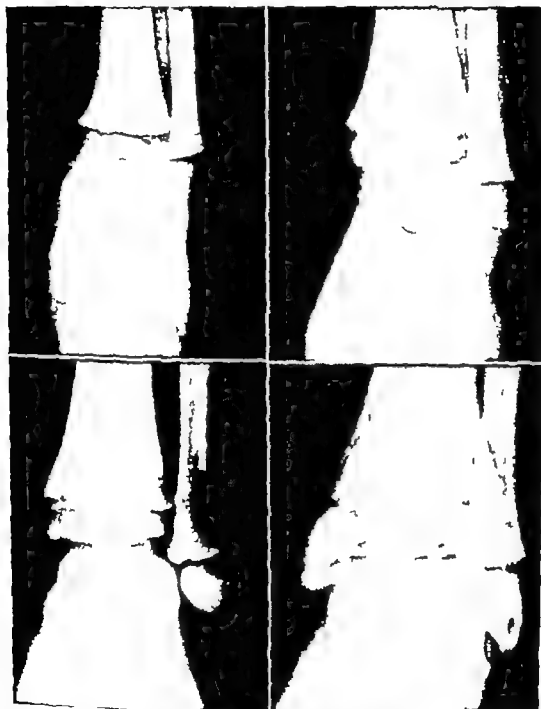


FIG. 12. The tibial malleolus. (A *Top left*) Tibiofibular socket just before the second year of life. (B *Top right*) The socket at 4. (C *Bottom left*) The ankle socket at 8 when bone begins to break down into the cartilaginous interior of the tibial malleolus. Note that at about this age the osseous core of the trigonal tubercle appears (Fig. 5 D). The trigonal tubercle moors another strong ligament, the posterior talofibular ligament. (D *Bottom right*) The tibiofibular socket at 12 when the tibial malleolus is ossified almost completely too, this age coincides with the fusion of trigonal center in the tail of the talus (Fig. 5 E).

The latter is nothing but scarred granulations that have failed to penetrate the distal fragment and have curled across the line of fracture giving the appearance of interposed periosteum. As over all epiphyses the periosteum enveloping the tibial malleolus is not such that it can be peeled and made to fall across the fracture line. It is thin and transfixed, it tears flush with the avulsed piece of bone leaving no loose tags. The torn edges of the deep fascia may fall between the fractured surfaces but the periosteum will not.

Interference with the blood supply of the detached distal fragment is regarded as the main cause of the common nonunion of avulsion fractures of the tibial malleolus. The transverse line of break, the limited area of medullary contact, faulty reduction and inadequate fixation all enlarge the vicious effect of the original vascular damage. The detached bone is deprived of its effective blood supply and may even undergo avascular necrosis. Since the tibial malleolus is not a weight bearing bone like the head of the femur and is not subject to rotary and shearing stresses as the broken fragment of the carpal navicular is even if completely deprived of its blood supply it does not crumble but retains its size and shape only

rarely does it manifest any evidences of "cystic" invasion or creeping replacement. In x ray films the detached malleolus may appear denser than the surrounding bone, but most often the contrast is not marked, for the comparison is made with the shadows cast by thicker bones.

The tibial malleolus is considered as important since it moors the deltoid ligament, which in turn suspends the calcaneonavicular or spring ligament of the longitudinal arch. The spring ligament consists of a fibrocartilaginous hammock slung across an angular gap medially between the os calcis and the navicular bone. The head of the talus rests on it. When the deltoid ligament loses its anchorage proximally the spring ligament sags and fails to support the head of the talus, the keystone of the longitudinal arch of the foot, the head of the talus sags, the longitudinal arch collapses and traumatic flatfoot intervenes.

In the treatment of avulsion fractures of the tibial malleolus, due respect must be given to its function as a suspension hook to the deltoid ligament. It is unwise surgery that advocates stripping of the ligament from the inferior border of the malleolus in preparing to introduce a screw or a bone graft upward. Metal pins can well be inserted



FIG. 13 The fibular malleolus (A Left) Freshly cleared specimen showing numerous nutrient foramina in the fossa just above the tip of the fibular malleolus. Note the paucity of nutrient apertures along the inferior border of the tibial buttress. (B Right) Fibular malleolus with an accessory epiphysis bipartite malleolus

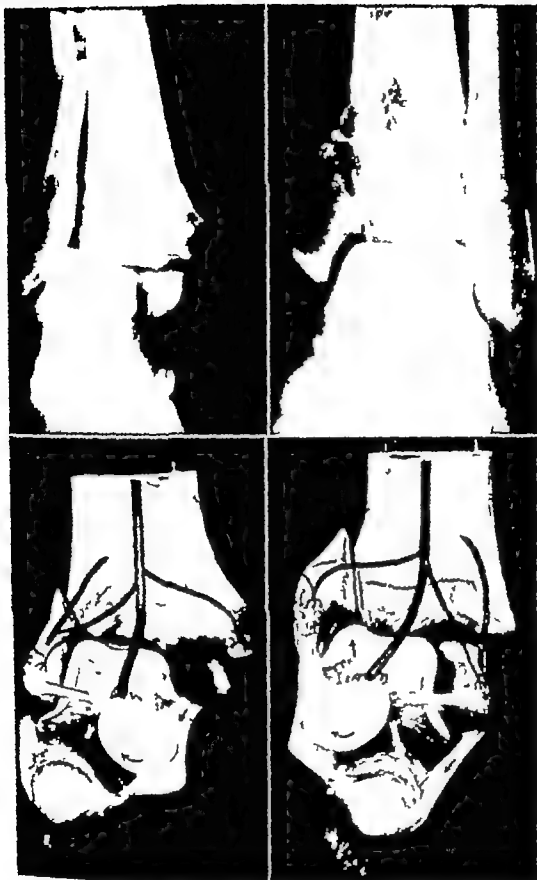


FIG. 14 Vascular relations of the two main types of tibial malleolar fractures. (A, *Top left*) Anteroposterior view roentgenograms of an avulsion fracture of the tibial malleolus. (B *Top right*) Anteroposterior view roentgenogram of a compression fracture of the tibial malleolus. The broken fragment is large and has had extensive area of medullary contact. (C, *Bottom left*) Drawing to illustrate the vascular relations of avulsion fracture of the tibial malleolus. The line of break falls at or below the level of penetration of nutrient vessels into the bone. (D *Bottom right*) Drawing to illustrate the circulatory relations of compression fractures of the tibial malleolus. Here the line of break passes above the medial to the level of penetration of nutrient vessels into bone.

through the malleolus without detaching the ligament, and bone grafts are best doweled from above. Onlay grafts are not advised, since the malleolus underlies the skin and offers gliding grooves to the tendons. Should

the malleolar fragment be too small to hold a pin or a screw, it is removed, and the split half of the *tibialis posterior* tendon is utilized to restore the suspensory function of the deltoid ligament (Figs 12-16).



FIG 15 Avulsion fractures of the tibial malleolus with signs of vascular damage. (A, *Top left*) A 3-month-old transverse fracture of the tibial malleolus with nonunion. Note the density of the malleolar fragment. (B, *Top right*) A year-old transverse fracture of both malleoli due to side-to-side shearing injury. The tibial malleolus is dense here an apparently avascular malleolar fragment has effected a union with the live bone above. (C, *Bottom left*) A year-old avulsion fracture of the tibial malleolus with redisplacement. The tibial malleolus shows evidences of creeping replacement (cystic degeneration) along its fractured side. The patient was allowed to walk under the illusion that the tibial malleolus had effected a fibrous union and this was considered to be sufficient—a common misconception. (Bottom right) Avulsion fracture of the tibial malleolus complicated by infection. Note the density of the malleolar fragment.

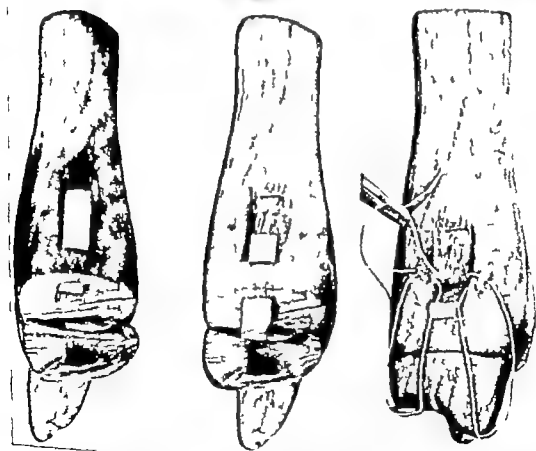


FIG. 16 Dowel graft for old ununited avulsion fracture of the tibial malleolus.
 (A, B C, Top) Wood models illustrating various steps of dowel bone graft.
 (D Bottom left) A 6-month-old ununited fracture of the tibial malleolus.
 (E, Bottom right) The same case 4 months after dowel bone graft

Compound Injuries and Soft Tissue Damage. The integument enveloping the ankle is tight it is comparatively immobile and close to the articular cavity. Over the malleoli it is thin, poorly nourished and vulnerable. A direct blow will compress the skin against the bone and cause it to slough, or tension will split tissues and expose the underlying joint. Not infrequently the ends of the bones are levered out through the skin. Compound injuries of the ankle are not uncommon.

No muscles except the vestigial fibers of the peroneus tertius cross the ankle. Since gas bacillus must have devitalized muscles to thrive upon, gas gangrene is not a frequent complication of compound injuries of the ankle. Numerous tendons cross the ankle and tendons like articular cartilage are practically avascular. Wounds heal sluggishly over exposed tendons. The quickest way to stop drainage from the ankle is to resect the tendons in the pathway of pus and to denude the articular ends of the bones of hyalin cartilage and promote ankylosis.

Skin ulcers which result from crushing ankle injuries are ragged in the edges, unhealthy at the base. In fractures produced by indirect violence at times the point gapes open through a transverse slit on the side of tension. The wound thus caused is clean cut, as if it had been caused by a knife. It heals favorably. Often in dislocation the skin sloughs secondarily due to pressure by the displaced bones. This is to be regarded as necrosis of the skin caused by compression from within. Healing is slow in crushing skin injuries.

Skin defects over the malleoli especially the tibial malleolus heal sluggishly. This is due chiefly to meager arterial supply but venous stasis plays a role as well. Superficial veins of the leg, the saphenae cross the area, and venous incompetence causes damming back of blood into the lower reaches of the limb. Seepage into the wound results in maceration and retards healing; infection produces induration of the base and

the margins of the ulcer. The superficial lymphatics of the leg accompany the saphenae and when the veins are infected the areolar tissue around becomes matted, and the lymphatic pathways undergo obliteration. Tissue fluids cannot pass proximally. In long-standing stasis the skin the fascia and even the bone thicken. There may be exuberant periosteal deposits and the interosseous membrane between the tibia and the fibula may undergo osseous metaplasia. Thickened, unyielding crural fascia constitutes a veritable barrier between the subcutaneous space outside and the muscular compartments within its confines. Indurated deep fascia fails to transmit the beneficial impulse of muscular contractions to the veins and the lymphatics under the skin, and stasis is aggravated. Segmental vein ligation and extirpation of thickened crural fascia will encourage the healing of these ulcers.

It is best to keep the wounds over the malleoli dry and clean. In compound injuries skin loss must be replenished as soon as possible. Preliminary to skin graft, superficial veins are ligated higher in the leg to obviate congestion, backflow and phlebitis. Avascular thickened portions of the deep fascia must be removed and split thickness skin graft applied as temporary covering. Thin grafts may take but they adhere to the bone, the tendons, the nerves and the arteries and cause pain and trophic changes; they break eventually due to friction from the shoes. In time they need to be replaced by cross-leg pedicled flaps bearing adequate padding of fat and fascia. In the aged, cross leg flaps entail considerable difficulty and are not without hazard. Compromise measures must be instituted. It is possible to mobilize adherent scars and pad them by bits of free fat or to underline them by a fat-bearing fascial flap either free or pedicled (Figs. 17, 18 and 19).

Sprains, Extravasation of Blood and Angiospasm. The tibial collateral or deltoid ligament is broad. It is continuous and confluent with the joint capsule. It is thick and strong. It pulls the attached malleolus off

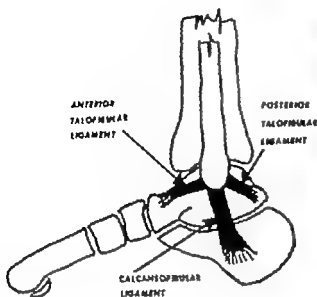


Fig. 9 Lateral hinges of the ankle (after Hughes)⁴

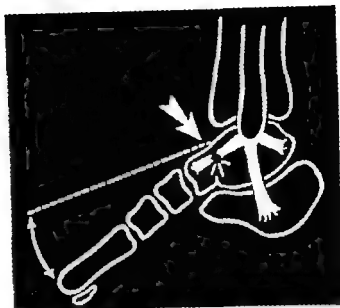


Fig. 10 The most common ankle sprain is a tear of the anterior short hinge occurring in equinus and inversion

accident room at the hospital. There examination reveals a somewhat overweight, slightly knock-kneed girl, holding her painful knee at approximately 15° flexion due to active muscle spasm. The internal lateral ligament is tender to pressure. The knee is otherwise stable, has no excess synovial fluid and roentgenograms are negative.

In checking the history of the patient, it develops that the girl has not injured her knee, that she can recall, but only recently has started to wear high heels exclusively. The previous evening had been spent in continuous dancing while wearing the high-heeled shoes. Then a diagnosis of an acute traumatic arthritis due to repeated minimal trauma can be made.

Dramatic relief often results from the use of ethyl chloride on the skin covering the internal lateral ligament. Placing the injured knee between two chairs (Fig. 8) the painful hump is sprayed with ethyl chloride for approximately one minute. The muscle spasm in the hamstrings then relaxes and the knee is allowed to come into complete extension. The pain leaves and then the patient is able to move the knee actively. Next, the knee should be rested in a plaster walking cylinder for 7 to 10 days, after which time quadriceps



Fig. 11 Maximum instability of the ankle mortise results from a tear of all three hinges (after Hughes)⁴

exercises are given. The patient should be cautioned concerning the advisability of acquiring an adequate sense of balance when wearing high-heeled shoes.

In the osteoarthritic knees, minimal overloads often produce an acute synovitis which, prior to the availability of hydrocortisone, was difficult to treat.

Ankle sprains are the most common athletic injury. They vary in severity according to their post-traumatic anatomy. There are 3 principal lateral ligaments of the ankle—2 short and 1 long (Fig. 9)—of which any combination of one or more may be torn.

The most common ankle sprain is a tear of the anterior short hinge (Fig. 10). This

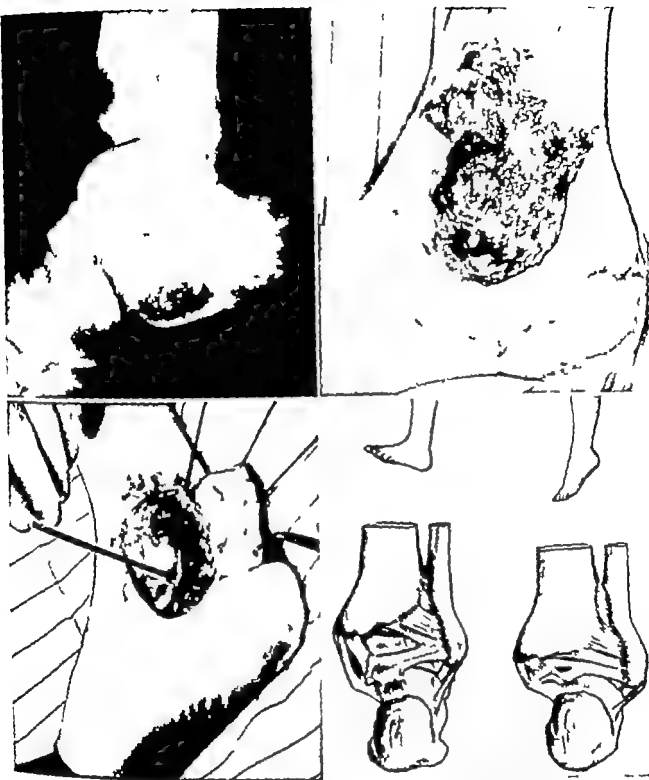
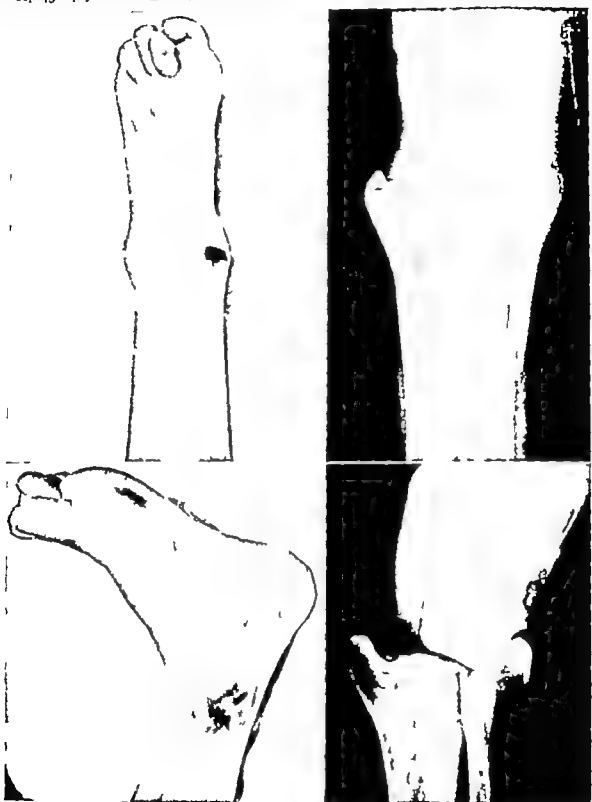


FIG. 17 Compound fracture-dislocation of the talus with skin slough. (A Top left) Old fracture-dislocation of the talus which had been reduced by manipulation and direct pressure notwithstanding the precarious skin over the displaced body of the talus. This roentgenogram was taken 2 months after reduction note the narrowing of subtalar joint space due to infection. (B Top right) Photograph showing extensive area of postreduction skin slough. (C Bottom left) After a prolonged course of chemotherapy the surgeon resected the ulcer and covered the area with split-thickness skin graft. The latter took but gave rise to a severe burning sensation in the foot. The split-thickness skin graft was lifted. The posterior tibial artery was missing, and the nerve (hooked with a probe) was found adhering to the skin graft. The chain of catastrophes might have been obviated had the surgeon slit the tense skin and extracted the displaced body of the talus. (D Bottom right) Drawing showing the avenue of extraction of the fractured, displaced body of the talus. When the foot is plantarflexed posterior talofibular and tibiofibular ligaments approach like the blades of scissors. In dorsiflexion the ligaments gape apart and bring the body of the talus into view. This avenue is also the best for draining infected ankles, as it is dependent. During surgery the foot should be placed in dorsiflexion.

FIG 18 Malleolar fractures with skin necrosis. (A, *Top left*) Fracture of the fibular malleolus with tear of the deltoid ligament. (B *Top right*) Medial view photograph of the same ankle. Note the ulcers and the surrounding discoloration. (C *Bottom left*) Fracture of the fibular shaft and tear of the tibiofibular and the deltoid ligaments (Dupuytren's fracture) (D *Bottom right*) Frontal view photograph of the same ankle showing the area of necrotic skin.



rather than tears across its fibers. When it rips through its own substance, it opens a rent into the articular cavity and causes hemarthrosis. The fibular collateral ligament is triadate; its cords are set off the joint capsule. Tears of the lateral collateral ligament are not accompanied by hemarthro-

sis but blood oozes into the subfascial space and causes discoloration of the webs of the outer toes and the lateral border of the heel.

The classical sprain of the ankle common in women involves the frontal component of the fibular collateral ligament. The anterior talofibular cord is a weak band; it connects

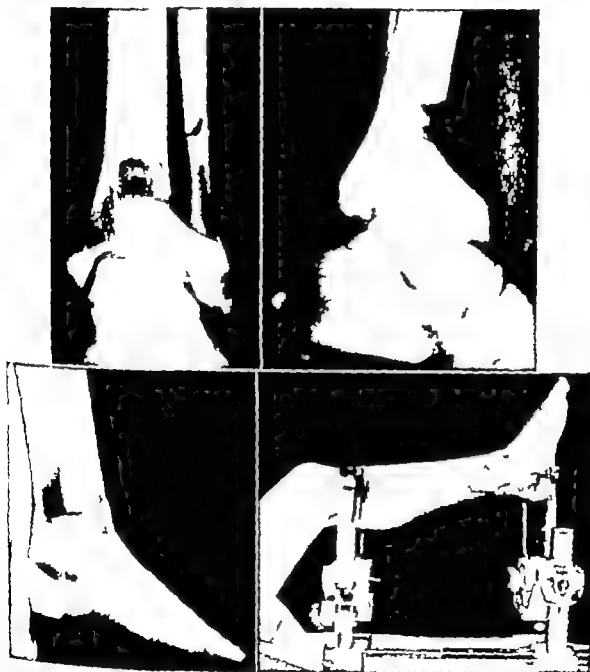


FIG. 19. Fracture of the bearing surface of the tibia with skin necrosis. Early reduction with 2-pin traction, without manipulation or surgery, is imperative in comminuted fracture of the ankle socket. (A, Top left) Comminuted fracture of the tibial vault in a man who fell from a second story and landed on his heel. (B, Top right) Lateral view of the same case. (C, Bottom left) Photograph of the ankle, showing large blisters. (D, Bottom right) Photograph of the ankle in a reduction apparatus. The ankle is reduced by traction and leverage through the os calcis pin, avoiding manual compression and kneading, hitches and wrenches which will further jeopardize the circulation of the skin.



FIG 20 Segmental vasospasm. (A, Left) Lateral view photograph of a 16-year-old girl who had undergone panastagalar arthrodesis. This was followed by extensive skin slough. The patient suffered from deep burning pain in the foot which was ascribed to involvement of the musculocutaneous branch of the peroneal nerve. (B Center) The same case after neurectomy of the musculocutaneous nerve and cross-leg skin graft. The deep burning pain in the foot persisted. Because of the surgeon's failure to palpate the pulsations of the dorsalis pedis, the patient was subjected to lumbar sympathectomy. (C, Right) Arteriogram following injection of contrast material into the femoral artery in Hunter's canal. Note that both tibial arteries are visualized. The dorsalis pedis is thin and threadlike. This patient was relieved completely after resection of the dorsalis pedis artery which was found imbedded in scar tissue.

the anterior border of the fibular malleolus with the lateral side of the neck of the talus. As the fibular malleolus is narrow from front backward and is placed posteriorly the ligament spans an appreciable gap on the anterolateral aspect of the ankle. When the foot is at a right angle to the leg, the fibers of the anterior talofibular ligament assume a horizontal direction and are relaxed. The ligament is tense in plantar flexion of the foot. Inversion, which usually accompanies plantar flexion, tightens the ligament. In this position, as with women who wear high heels a slight misstep will twist the foot farther inward and sprain the ligament. In plantar flexion when the narrow posterior body of the talus glides forward within the widening malleolar recess in front, there is room for the talus to be swayed from side to side and the short tibial malleolus on the other side allows the foot to be carried more medially. Moreover the anterior talofibular ligament receives practically no support

either the tendons or the fascial bands. It is crossed in front by the tendon of the peroneus tertius which is weak compared with the stout tendon of the tibialis anterior that braces the area in front of the tibial malleolus. The anteromedial corner of the ankle receives additional protection from the upper branch of the Y-shaped inferior extensor retinaculum. The stem of this retinaculum starts from the lateral aspect of the os calcis below and just misses the area spanned by the anterior talofibular ligament. Besides the tendon of the peroneus tertius the anterior talofibular ligament is crossed by the perforating branch of the peroneal artery and the musculocutaneous nerve. Tears of this ligament are accompanied by considerable extravasation of blood and pain. Whether the pain in these sprains is due to vasospasm, to nerve irritation or to increased tonus of the peroneus tertius is a matter of conjecture. It is relieved by regional infiltration of procaine. Since the deep fascia thus

out in front of the fibular malleolus and there is laxity of tissues in this region, sprains of the anterior talofibular ligament often are accompanied by considerable edema and puffiness. Pressure dressing in the form of elastic bandage is in order. Even in complete tears of the anterior talofibular

ligament the stability of the ankle is not affected, and prolonged immobilization is hardly warranted. To avoid recurrence the patient is advised to wear shoes with stable heels. In the less common tears of the middle cord of the fibular collateral ligament, the stability of the ankle is affected and in



FIG. 21 Osteochondritis. (A Top left) Osteochondritis dissecans. (B Top right) Ulcerative chondromalacia (C Bottom left) Osteochondritis tili (D Bottom, right) Lateral view of the same case.

sprain-fractures of this component the bony piece fails to unite. The ankle must be immobilized adequately; fibrous union obtained by continued rest will help to stabilize the ankle.

In all surgical procedures about the ankle except gravitational ulcers and compound injuries where one has no choice, it is wise to place the incision along the longitudinal axis of the limb and to avoid undercutting; the skin incision should cut flush through the deep fascia, and if flaps have to be developed, the undermining should be done as near the skeletal plane as is feasible. Incisions should not cross the arterial trunks because with subsequent scarring, spasm or stenosis of the artery may give rise to intractable pain (Fig. 20).

Osteochondritis and Osteocartilaginous Loose Bodies. The angle formed by the superior articular surface of the body of the talus and its medial malleolar facet—the point highest and farthest away from the outer neck where most vessels enter the bone—constitutes the most common site of osteochondritis dissecans involving the ankle joint. Occasionally a cartilage-coated seed

of bone will flake off from the outer border of the trochlea tali and rarely from the tibial surface. In all these instances osteochondritis dissecans appears to originate at or near the junction of two articular facets. As with the femoral head, osteochondritis sometimes affects the entire superior bearing surface of the body of the talus.

Completely detached loose bodies usually are found in the anterior or the posterior chambers of the ankle where there is room. A solitary seed in the posterior compartment may be confused with os trigonum or fractured trigonal process. In arthrograms the shadow cast by air introduced into the ankle will surround the osteocartilaginous body; the shadow will settle above the os trigonum but will neither surround nor pass under it. In fractures of the trigonal tubercle the ankle and the posterior talocalcaneal joints communicate through the crack in the tail of the talus, and air will sandwich itself between the separated bones or will pass from the talocrural into the posterior talocalcaneal joint. Rarely does an osteocartilaginous body appear in the small pouch below the tibial malleolus when it does it

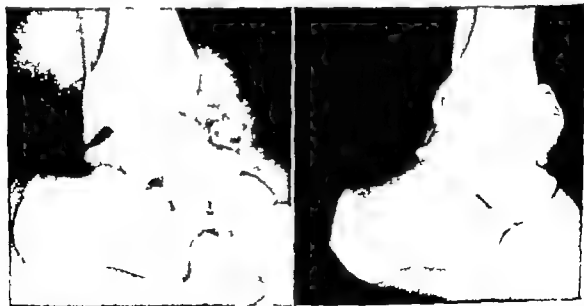
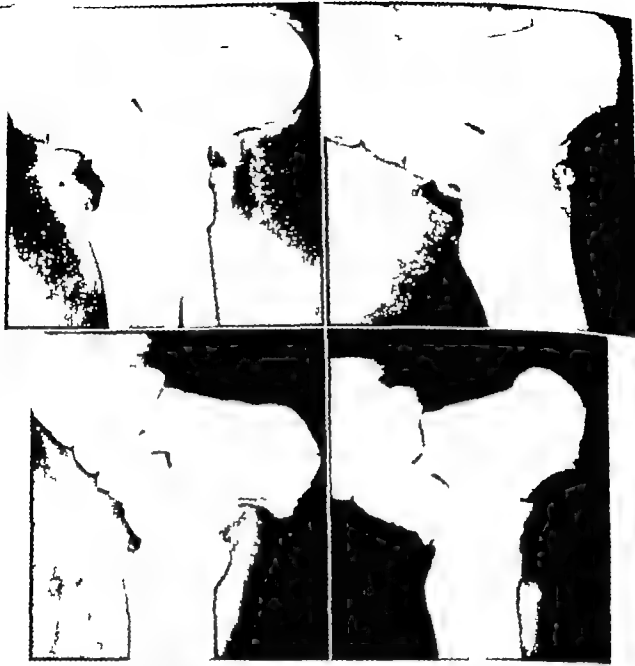


FIG. 22. Osteocartilaginous loose bodies in the anterior compartment. (A Top) Arthrogram showing osteocartilaginous loose bodies in the anterior chamber. These are possibly of synovial origin, as the trabeculae appear to be coarse and lack definite architectural design. (B Bottom) Large osteocartilaginous loose bodies in the anterior chamber. Note that these bodies are finely trabeculated; they arose most probably from the mother bone.

It is advisable to extract osteocartilaginous loose bodies floating freely in the joint cavity. Partly detached osteochondritis dissecans also is removed surgically. The defect left above the articular surface from which the osteochondritic body had loosened is shaved off down to bleeding bone and smoothed out (Figs 21 22 and 23).

It is regarded as an avulsion ossicle. The detached ligament is confluent with the joint capsule, and the piece of bone it pulls off will line the joint cavity. The fibular collateral ligament on the outer aspect of the ankle sets off the joint capsule, avulsion ossicles below the fibular malleolus are extra articular

FIG. 23 Osteocartilaginous loose bodies in the posterior chamber (A Top left) Arthrogram of the normal ankle (B Top right) Arthrogram in a patient with os trigonum. Note that the shadow cast by air sits on the os trigonum (C Bottom left) Arthrogram where the air shadow surrounds an osteocartilaginous loose body (D Bottom, right) Arthrogram in an old fracture of the tail of the talus. Here the shadow cast by air sandwiches itself between the broken tail and the main body of the talus



SUMMARY AND IMPLICATIONS

1 The terminal divisions of the popliteal artery form a complete loop around the ankle, when one arterial trunk is injured or blocked the other will supply the foot adequately

2 The proximal and the distal poles of the great arterial loop are caught between bones and in front of the ankle the anterior limb of the loop is crossed by the long extensor tendon of the big toe the extensor hallucis longus tendon and its fascial retinaculum build a hedge along the medial aspect of the dorsalis pedis and in outward dislocations of the foot carry the artery laterally. Thus the anterior limb may be displaced and even compressed but it seldom is torn. On the other hand, the posterior limb sometimes is torn in dislocation fractures of the talus with the body fragment completely extruded from the posteromedial corner of the ankle socket the displaced bone pins the artery against the unyielding flexor retinaculum

3 In transcervical fractures of the talus, when the body fragment is dislocated it usually dies avascular necrosis is also certain in fractures of the body of the talus when the line of break cuts across two cartilage coated facets

4 Vascular damage is behind nonunion of avulsion fractures of the tibial malleolus.

5 The skin in front of the ankle is poorly nourished. The tendons and their fascial retinaculae intervene between the arterial network and the integument sizable arteries cannot reach the skin due to frictional interference by gliding tendons. Wounds in this area heal sluggishly. Moreover the integument above the tibial malleolus and the inner surface of the distal tibia overlies the long saphenous vein venous backflow causes seepage and maceration of the tissues, regional phlebitis and skin sloughs. Vein liga-

tion and extirpation of the crural fascia should supplement orthopedic and plastic measures

6 No muscles—except the vestigial peroneus tertius—cross the ankle. Gas gangrene is not a common complication of compound injuries of the ankle

7 Numerous tendons cross the ankle and traumatized tendons—like cartilage-coated detached bone or thickened scarred fascia—are avascular. They should be suspected as the cause of protracted drainage

8 The perforating peroneal artery passes over the front of the anterior talofibular ligament and arterial spasm may be behind the inordinate pain which often is associated with sprains of this ligament segmental stenosis of the anterior tibial and its continuation the dorsalis pedis artery may be responsible for the intractable pain which at times accompanies incisional scars in front of the ankle

9 Osteochondritis dissecans and osteo-cartilaginous loose bodies of the ankle are not uncommon and here again the blood vessels nourishing the loosened pieces must have been blocked or severed.

10 Since the ankle is primarily a weight bearing joint, in all surgical undertakings stability must take precedence over movement there should be no hesitancy to eliminate painful joint movement by arthrodesis in cases of avascular necrosis of the broken bony components of the ankle as in arthritis due to any other cause

BIBLIOGRAPHY

- Kelikian, H. Gravitational leg ulcers, *J. Internat. Coll. Surgeons* 15 111 128 1952.
 ———. Dynamic and circulatory relations of the ankle. *Am. Acad. Orthop. Surgeons Instructional Courses*, Vol. 9 Chap. 21 pp. 347 360 1952

Treatment of Femoral-Neck Fractures

RICHARD P. GILBERTY, M.D.*

The treatment of fractures of the femoral neck must necessarily vary in accordance with the conditions found associated with each individual case. An attempt will be made in this paper to correlate the significant factors that have to be considered and the influence that they exert on the final outcome of the fracture.

The problem of nonunion and aseptic necrosis is ever present. The fracture committee of the American Academy of Orthopedic Surgeons reported that about 30 per cent of all fresh femoral-neck fractures end in nonunion. Of the fractures that do unite approximately 40 per cent develop aseptic necrosis. This means that only 30 per cent are satisfactory.

The terms aseptic necrosis and degenerative arthritis are sometimes used interchangeably. Aseptic necrosis per se has been defined as changes in which there is x-ray evidence of various densities in the femoral head, with collapse of the head. Degenerative arthritis is present when there is x-ray evidence of loss of joint space and acetabular changes, and also with density changes of the head. In both instances there is a period of functional use followed by a progressively painful hip.³

Following union of a fracture of the femoral neck, a period of 18 months to 3 to 4 years may elapse before clinical and x-ray findings of aseptic necrosis will be present.

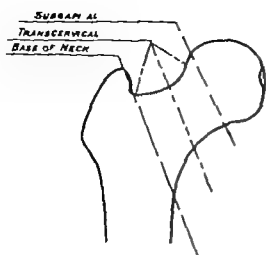
An accurate evaluation of end results can be ascertained only if at least 4 years have elapsed since the date of the original fracture.

Fractures of the femoral neck occur in children, and unfortunately the end results are not always favorable. Aseptic necrosis does occur and has been reported to be as high as 40 per cent.¹⁴

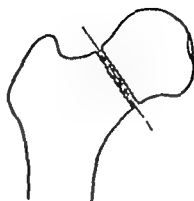
The ossifying center in the developing head of the femur in infants and in children under 10 receives its blood supply from the visceral capsule vessels. The ligamentum teres vessels do not enter the head of the femur in children nor do they contribute to the nourishment of the growing femoral head. The anastomosis between the ligamentum teres vessels, the capsular arteries and the nutrient artery of the shaft does not take place until ossification of the head of the femur is practically complete.¹⁵ Almost all writers agree that the neck vessels in a child do not penetrate the epiphyseal plate to reach the femoral head.⁷

When ossification of the head of the femur has occurred, the anastomosis between the foveal vessels, the capsular arteries and the nutrient artery of the shaft is complete. This does not simplify the problem of femoral-neck fractures in the adult. The majority of the capsular arteries are posterior and only a few branches are found in the anterior portion of the capsule. In an intra-capsular fracture the ensuing hemorrhage causes marked distention of the joint capsule. When a fresh fracture is opened through an incision of the anterior capsule,

* From Orthopedic Service, Nassau and Mendbrook Hospitals.



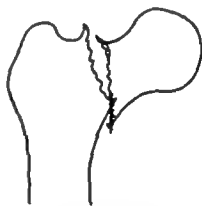
LEVELS OF INTRACAPSULAR FRACTURES



TRANSVERSE FRACTURE IDEAL FOR INTERNAL FIXATION



COMMINUTED FRACTURE NOT AMENABLE TO ANY TYPE OF FIXATION



OBLIQUE FRACTURE: SHEARING FORCE TOO GREAT FOR ANY METHOD OF FIXATION

FIGURE 1

the welling-up of blood in the wound causes the surgeon to recognize the tension to which the capsule has been subjected. It necessarily follows that with relief of this tension the pressure is taken off the capsular arteries and in effect must improve the blood supply which had been embarrassed. This is

in contradistinction to the ideas of many surgeons who hesitate to open a femoral neck fracture fearing this would further impair the capsular blood supply.

Opening a fracture of the femoral neck has other advantages. Besides shortening the operating time it enables the surgeon to



FIG 12 Inversion roentgenogram demonstrating instability of the ankle mortise.

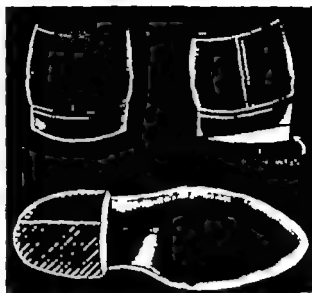


FIG 13 Lateral wedge of heel for convalescent sprains.

particular tear occurs in equinus and inversion. Variable degrees of ankle mortise instability result, depending upon the extent of the damage. When rupture of the long hinge is added to rupture of the short hinges (Fig 11), maximum ankle mortise instability results.

An unstable ankle mortise^{4,6} can be demonstrated by roentgenograms of the ankle with the foot in inversion (Fig 12). Varying with individual injuries, anesthesia may even be required to conduct such an examination.

At first glance, the present methods for treatment of ankle sprains would seem to be at variance with one another. The following treatments are practiced at the election of the physician: (1) elevation, ice packs, and crutches; (2) taping; (3) massage, active motion, and taping (athletic trainer); (4) procaine block and active weight bearing (Leriche) and (5) walking cast.

Elevation, ice packs, and crutches are discarded as being unsound economically. A patient cannot afford to put up with them. Taping is one of the worst forms of treatment because most physicians tape an ankle that has been sprained as they would if the individual were going to play football all afternoon. By this is meant that an extensive occlusive circular dressing is applied. The

ankle swells in a few hours and the tape becomes an excruciatingly painful tourniquet. The smart patient will tear off the tape. The ever-trusting patient, aided with some codeine, may go on to a serious circulatory disturbance. Taping is not adequate to control secondary swelling.

The athletic trainer accomplishes his results by devoting more time to his patient than physicians are willing to give. "Doc" James O'Reilly of the Kansas City Athletic Club, advisory coach for several Olympic teams, tells me that the trainers will work all night with an ankle sprain, icing and massaging until pain is free, keeping the ankle moving, and then taping it. An ankle may be taped two or three times the first night to keep ahead of any swelling. As a result, secondary swelling is combated successfully and the athlete is able to compete on the following Saturday.

The procaine injection method of Leriche^{4,6} is becoming increasingly popular and, at first glance, would seem to be diametrically opposed to the walking cast method.

Successful treatment by whatever method elected accomplishes one common objective, i.e., control of secondary swelling. The skilled trainer controls secondary swelling by technical loving massage, gentle motion, and ac-

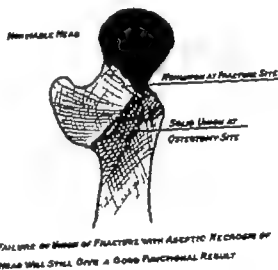
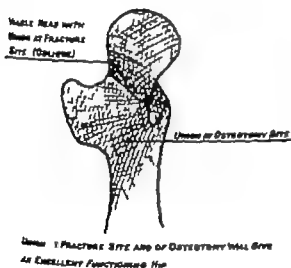
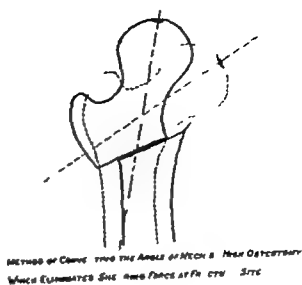
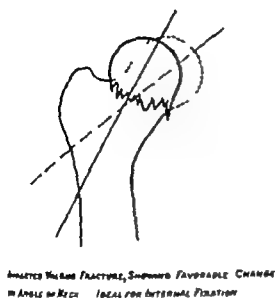


FIGURE 2

determine accurately the type of fracture present and to ensure a more nearly accurate reduction.

In a child the cartilage of the femoral head receives its nourishment from the synovial fluid. In adults it is dependent upon the subchondral vascular network for its nutrition. When there is circulatory embarrassment over a prolonged period of time such as that which may follow a fracture of

the neck, the cartilage degenerates and is no longer able to recuperate.

In the older age groups other factors which must be considered, in that they play an important part in the treatment of this fracture are the changes which result from the process of aging. There is a normal tendency for extensive necrosis of the cells of the cortical interstitial lamellae to occur in older individuals and this process pro-

gressively increases with age.¹⁰ The vascular nourishment of the head of the femur varies with the physiologic development of the individual and with the extent of the involution of aging. The changes resulting from the relative anoxemia are manifested by a diffuse atrophy affecting all the elements of bone structure and a focal form of atrophy with localized cavitations within the trabecular structure.⁸

In the articular ends of long bones, there normally is present marrow which grossly had a reddish color containing only a very small proportion of fat. In the shafts of long bones, the marrow is yellow and contains a large amount of fat (96%). In the older age groups, the head of the femur on section shows a variance in that there is a decided change in appearance in contrast with young adults. Instead of the reddish appearance found in the young it has a yellowish to grayish color signifying the presence of large amounts of fat. For example a femoral head in a 20-year-old individual was removed and sectioned immediately and macroscopically it was found that the lamina system of the entire head presented a reddish color up to the articular cartilage. Sectioning of a head taken from a patient 52 years old showed a similar pattern—reddish in color—but there were several small scattered areas of yellowish discoloration. The articular cartilage was white and glistening. As heads were sectioned immediately after removal in older age groups from 70 to 90 it was found that there was a marked substitution for the reddish lamina by yellowish to almost grayish discoloration. Similarly the cartilage of the head showed marked loss of the normal glistening smooth surface found in younger adults. All the heads in the older age groups were removed following acute fractures. In none of these cases was there x ray evidence suggestive of pre-existing advanced arthritis. Therefore, it may be concluded that these changes are a result of the normal process of aging, which may be directly due to diminished blood supply. Recently, Trueta¹¹ made a similar observa-

tion of sectioned femoral heads obtained from adult cadavers. He noted that the entire epiphysis was occupied by yellow marrow except for a small area underlying the fovea capitis, only the metaphyseal portion having red marrow.

Interference with normal blood supply to bone impairs physiologic processes and causes bone to degenerate. Chandler⁴ performed microscopic studies on femoral heads and found marked alteration in the cellular elements when there has been either complete or incomplete interruption of the blood supply. There was absorption of the bony trabeculae and the marrow spaces were filled with necrotic material and fibrous tissue. Microscopic sections were made of a femoral neck fracture in which the proximal portion was removed 1 week after the injury. The patient R. K. was 55 years old and suffered from Parkinson's disease. These sections revealed spicules of degenerated to necrotic bone. Microscopic studies are being made of a series of femoral heads removed within 24 to 48 hours after fracture in the older age group. These findings will be reported in a subsequent paper.

Vessels from the medial femoral circumflex artery supply four fifths of the head of the femur.¹² However they are traumatized easily being most vulnerable in displaced fractures. If they are contused severely and possibly are torn, there will result a complete shutoff of blood supply to the proximal fragment.

Following a displaced subcapital or mid-neck fracture of the hip in the elderly patient, the proximal portion virtually becomes a sequestrum. Further damage to an already impaired blood supply is sufficient to cause the entire head to degenerate. Although this is not recognized by x ray findings immediately it becomes quite definite after a period of time has elapsed. When the condition is recognized, the most that can be expected with prolonged treatment, which involves the use of bone grafts etc. is what is described by Phemister¹² as the gradual replacement by creeping substitution for the

dead trabeculae and by the ingrowing of vessels which bring with them embryonic fibroblasts which are capable of new bone formation. This may heal the fracture well in certain cases, but the pre-existing conditions that have resulted from the prolonged period of impaired blood supply will not be altered.

Occasionally a fracture occurs in which the proximal fragment actually is impacted in the distal portion; this is the so-called valgus or abduction type of fracture. There results an interlocking of the lamina of the internal weight bearing system as described by Farkas et al.⁶ This system has a rich endosteal lining and together with the absence of motion at the fracture site causes the fracture actually to go on to a rapid bony union even in the older age groups. There is less tearing and confusion of the capsular vessels in this type of injury. The force is spent telescoping the 2 bone fragments rather than abutting on adjacent soft tissue.

Intracapsular fractures are classified as to the level of the fracture and also as to the type. The level is designated as to whether the fracture line extends through the subcapital region, i.e., at the junction of the head and the neck, through the mid-portion of the neck or through the base of the neck; the type as to whether it is comminuted, transverse or oblique.⁶

It must be remembered that the muscles about the hip exert considerable force and, depending upon the type of fracture sustained, the component of this force may be either beneficial or detrimental. With a transverse fracture the vector of force acts to compress the 2 fragments; when the fracture is oblique to the neck or when there is comminution the vector is a shearing force. The force of compression is favorable in that it provides for easier fixation and may even promote osteogenesis. In the oblique fracture the shearing force is so great that regardless of the attempted method of fixation the majority of them open up and go on to nonunion.

Taking all of these factors into consideration, our group has been instituting the following methods in the definitive treatment of femoral neck fractures.

In children our experience has been limited by the rather infrequent occurrence of this fracture. However, because of the poor results that have been obtained with conventional treatment, a primary osteotomy is the method of choice in all fractures where Pawles angle is greater than 50° as advocated by Allende and Lazama.¹

In adults, base-neck fractures which are not comminuted or oblique are treated by internal fixation using a Smith-Petersen nail with a plate Jewett or McLaughlin type. This method has been satisfactory with this specific type of fracture.

Transverse fractures through the mid-portion of the neck in which there is no comminution are fixed with Smith-Petersen nails under direct vision by opening the fracture; this ensures proper placement of the nail and also an accurate reduction. Impacted valgus fractures are treated similarly.

When the fracture is found to be comminuted or oblique an immediate osteotomy is performed rather than attempting to reduce the fracture and use some method of internal fixation. This will ensure a good result in a greater percentage of patients primarily. If in the event that the articular cartilage degenerates as mentioned previously or the fracture site fails to unite the osteotomy itself will minimize the disability. The loss of function resulting from a primary osteotomy does not approach that lost from a second procedure. Anker et al.² reported 32 femoral neck fractures treated by primary osteotomy in which only 9.3 per cent developed a clinical degree of aseptic necrosis.

Our group prefers a high McMurray type osteotomy. Best results are obtained when weight bearing is restricted until solid bony union of the osteotomy site has occurred. Motion at the osteotomy site is evaluated by taking roentgenograms with the extremity in extreme abduction and adduction. If this procedure is painless and both fragments

move in unison, it may be concluded that union is complete. In some cases the original fracture does not heal by bony union, but this does not alter the functional result, which is unusually good. In one patient, J. H., the osteotomy site united but not the fracture of the femoral neck. A McMurray type of osteotomy was performed in 1939 for this ununited femoral neck fracture of 1 year's duration. The following year he was able to return to his former occupation as a roofer. He continued to perform arduous work for a period of 10 years without any difficulty. Function of his hip was excellent, and he did not experience any pain.

Using this classification 147 femoral neck fractures were treated at Nassau Hospital (these included all types described above) between 1939 and 1947. Of these 113 or 76.8 per cent were considered a good end-result, 5 or 3.4 per cent, a fair result and 29 or 19.7 per cent were failures.

In a subcapital fracture of an elderly patient who is in fairly good state of health and can be rehabilitated, rather than performing an osteotomy many surgeons are excising the head and replacing it with a prosthesis. When the patient's general health precludes any possibility of rehabilitation, i.e. he is physically incapacitated or partially paralyzed, Hudson⁸ advocates excision of the head alone. In so doing, the patient no longer has to be subjected to the constant agony of pain from the fracture but instead to the relatively minor discomfort of a wound. This procedure definitely shortens the period of hospitalization and simplifies nursing care.

SUMMARY

1 The blood supply to the femoral head in children and in adults is vulnerable and becomes inadequate in many instances following fracture of the femoral neck.

2 Opening a femoral neck fracture does not promote further impairment of circulation and ensures a more nearly accurate reduction and evaluation of the fractures.

3 In the elderly patient, the prefracture status of the femoral head precludes bony union when there has been further impairment of the blood supply incident to the trauma.

4 Fractures of the femoral neck should be classified both as to the level—subcapital, transcervical or base of neck—and as to type—transverse, oblique or comminuted.

5 Primary osteotomy is the treatment of choice in oblique and comminuted fractures. Nonunion of the fracture but solid union of the osteotomy site does not necessarily produce a failure. A good functional and painless hip can be expected in most instances.

REFERENCES

- 1 Allende G., and Lezama, L. G. Fractures of the neck of the femur in children: a clinical study. *J. Bone & Joint Surg.* 33A: 387-395 1951.
- 2 Anker F. J. and Nelson M. D. Primary osteotomy for treatment of intracapsular fractures of the neck of the femur. *Ann. Surg.* 155: 69-78 1952.
- 3 Badgley C. E., and Denham, R. H. Aseptic necrosis of femoral head following fracture of hip. *J. A. M. A.* 137: 1193-1196 1948.
- 4 Chandler F. A. Observation on circulatory changes in bone. *Am. J. Roentgenol.* 44: 90-97 1940.
- 5 Dufour J. J. Pre fracture status of femoral neck. *Internat. Abstr. Surg.* 95: 585 1952.
- 6 Farkas, Aladar Wilson M. J. and Hagner J. C. Anatomical study of mechanics, pathology and healing of fracture of femoral neck. *J. Bone & Joint Surg.* 30A: 53-69 1948.
- 7 Howe, W. W. Lacey T. and Schwartz, R. T. Study of gross anatomy of arteries supplying proximal portion of femur and acetabulum. *J. Bone & Joint Surg.* 32A: 856-866 1950.
- 8 Hudson, O. C. The use of excision of the head in the treatment of fracture of the neck of the femur. *J. Kentucky M. A.* 3: 480 1952.
- 9 Hudson, O. C. Bartels, W. P. and Freese, C. F. Acute fractures of the femoral neck. *Am. J. Surg.* 81: 215-220 1951.
- 10 Jaffe and Pomeranz. Changes in the bones of extremities amputated because of arteriovascular disease. *Arch. Surg.* 29: 565-580 1934.

- 11 McNeur J C The treatment of sub-capital fractures of the neck of the femur with a nail plate and wedge osteotomy J Bone & Joint Surg 35B 188 191 1953
- 12 Phemister Dallas B Treatment of the necrotic head of the femur in adults, J Bone & Joint Surg 31A 55-65 1949
- 13 Trueta, J., and Harrison M The normal vascular anatomy of the femoral head in adult man J Bone & Joint Surg 35B 442 461 1953
- 14 Wilson John C Fractures of the neck of the femur in childhood J Bone & Joint Surg 22:531 546 1940
- 15 Wolcott Eugene W The evaluation of the circulation in the developing femoral head and neck Surg Gynec & Obst. 77 61 68 1943

Index

- Abduction hip joint, 101
 - wide strain from, 102
- Acetabular artery, 100
 - trauma in dislocation of hip joint, 106
- Acetabular foramen, 99
- Acetabular ligament, transverse anatomy, 99
- Acetabulum, fracture, with dislocation of hip joint, 102, 104
 - reduction, 103
- Acromioclavicular joint, dislocation, 10-12
 - diagnosis, roentgen-ray, 10-12
 - etiology, 12
 - treatment, 12
- Adduction, hip joint, 101
 - wide strain from, 102
- Adductor muscle(s) brevis, flexion of hip joint, 101
 - lateral rotation of hip joint, 101
 - laceration in dislocation of hip joint, 106
 - longus flexion of hip joint, 101
 - lateral rotation of hip joint, 101
 - magnus, ischial portion, extension of hip joint, 101
 - lateral rotation of hip joint, 101
 - pubic part, flexion of hip joint, 101
- Age as factor incidence giant-cell tumor of bone, 134
- Albee operation for simple dislocation of patella, 62, 64
- Amputation, giant-cell tumor of bone, 132
- Anderson, William, surgical technic for fracture of patella, 4
- Anesthesia, local, procaine, menisci, traumatic lesions, 44
 - nitrous oxide and thiopental sodium menisci, traumatic lesions, surgical treatment, 44
 - spinal, menisci, traumatic lesions, surgical treatment, 44
 - thiopental sodium with nitrous oxide, menisci, traumatic lesions, surgical treatment, 44
- Angospasm, sprain, ankle, 200 203 206
- Ankle joint, anatomy, 179
 - surgical, 109 111
 - dislocations, 188-190
 - fractures, avulsion, tibial malleolus, treatment, 196 198 199
 - vascular relations, 194-196
 - behavior, 186-190
 - comminuted, tibia, bearing plate, treatment, 193 194
- Ankle joint (Continued)
 - fractures (Continued)
 - compound with dislocation, 201
 - and soft-tissue damage, 200-203
 - compression tibial malleolus, 194
 - fibula malleolus, vascular relations, 193 194
 - tibia malleolus, vascular relations, 194
 - transcervical with dislocation partial treatment, 188 190
 - without dislocation treatment, 188
 - vascular relations, 190 192 199
 - attachments, ligaments, 193
 - behavior of broken bones, 193 198
 - extent of articular coating, 192
 - fulcra, 192 193
 - grooves, 192 193
 - growth pattern, 190 192
 - nutrient foraminae, 193
 - proximity of networks and laxity of intervening tissues, 193
 - ligaments dislocation, complete without associated fracture, 118 119
 - surgical treatment, 119
 - external (lateral) surface, 109 110
 - internal (medial) surface, 110 111
 - posterior surface, 112
 - rupture with avulsion of internal malleolus, 117 118
 - with disruption of ankle mortise, 115-119
 - surgical treatment, 115 119
 - vitallium screws, 115 118
 - physical examination, 111 112
 - types, 115 117
 - sprains, 109 115
 - complications, 17 18
 - from maltreatment, 119-120
 - arthritis, traumatic, 120
 - atrophy Sudeck's, 119-120
 - osteoporosis, 119
 - weakness of ankle, 120
 - definition, 109
 - physical examination, 111 112
 - treatment, 16-18 112 115
 - bandage, compression elastic, 114-115
 - gauze, 114
 - hyaluronidase injection, 114 116
 - massage, 16-18
 - Mercurochrome, 113

- Ankle joint (*Continued*)
 ligaments (*Continued*)
 sprains (*Continued*)
 treatment (*Continued*)
 plaster cast with walking heel 115
 procaine injection 113
 Leriche technic, 16 17
 splint, basket weave 113 115
 strapping with adhesive tape 113 114
 walking cast, 16-18
 tears 15 16
 mortise stable 18
 unstable 16 18
 movements, 179
 necrosis (avascular) in dislocations and fractures of tibia, 185 190
 attachments ligaments 185 186
 broken bones, behavior 186-190
 extent of articular coating, 185
 fukra, 185
 grooves 185
 growth pattern 185
 nutrient foraminae 186
 proximity of vascular networks and laxity of intervening tissues 186
 osteocartilaginous loose bodies 206-207
 osteochondritis 205 207
 physiology 109-111
 sprains extravasation of blood and angiospasm, 200 203 206
 fibular collateral ligament involvement, 203 206
 vascular relations arterial pathways, 179 183
 arterial loop accessory 180 183
 great, 180
 arteriograms, 181
 rete, lateral tarsal 180-181 183
 tibial malleolar 181 183
 roentgenograms, 182
 clinical correlation, 185 207
 angiospasm 200 204-206
 avascular necrosis in dislocations and fractures of talus, 185 190
 extravasation of blood, 200 203 206
 fractures, 190 192 203
 osteocartilaginous loose bodies, 206-207
 osteochondritis, 205 207
 sprains 200 203 206
 lymphatics, 183 185
 direction of flow in foot 183-184
 veins, 183 185
 direction of flow in foot, 183 184
 Arteriograms, arterial pathways, 181
 femoral artery in Hunter's canal, 204
 tibial artery tear and displacement, 191
 Artery(ies) acetabular 100
 trauma, in dislocation of hip joint, 106
 Artery(ies) (*Continued*)
 ankle joint, pathways, 179 183
 accessory loop 180 183
 arteriograms 181
 great loop 180 183
 roentgenograms 182
 tarsal rete lateral, 180-181 183 193
 tibial malleolar rete 181 183 193
 circumflex, femoral, medial, 212
 dorsalis pedis, 180
 femoral *See* Femoral artery
 foveal 100
 genicular medium 107
 gluteal. *See* Gluteal artery
 nutrient, 186
 fibular shaft, 190
 obturator 100
 peroneal 180 183
 plantar lateral, 180
 popliteal, anatomy 107 180
 injury 25 26
 pudendal internal capsular branches 100
 trauma, in dislocation of hip joint, 106
 tibial, anterior 180
 displacement, 191
 posterior 180
 tear 191
 Arthritides, diagnosis, differential, from menisci, traumatic lesions 43
 Arthritis, degenerative definition 209
 gonorrheal, diagnosis differential, from menisci, traumatic lesions 43
 hypertrophic with spurs from articular surfaces of tibia or femur diagnosis differential, from menisci, traumatic lesions, 43
 rheumatoid, low grade diagnosis, differential, from menisci, traumatic lesions, 43
 traumatic acute, knee 15
 of finger 9
 from maltreatment of sprains of ankle joint, 120
 with sprain ankle 18
 with spurs from articular surfaces of tibia or femur diagnosis, differential, from menisci, traumatic lesions, 43
 tubercular diagnosis, differential from menisci, traumatic lesions, 43
 Arthrodesis ankle fractures of talus, compound, with dislocation 190
 transcervical without dislocation 188
 with partial displacement, 188 190
 knee, step-cut fusion with medullary nailing *See* Knee step-cut fusion with medullary nailing
 Arthrography in diagnosis, rupture of rotator cuff of shoulder without dislocation or fracture 93 94

- Arthroplasty cup of Smith-Petersen dislocation of hip joint, 106
in knee operations use by early American surgeons, 4
- Arthrotomy "locked" knee removal of medial meniscus, 52
- Aspiration of effusion in dislocation of patella simple 61
in knee injuries, 25
of hemarthrosis, in dislocation of patella, simple 61
- Atrophy Sudeck's, from maltreatment of sprains of ankle joint, 119 120
treatment, 120
- Avulsion fracture of greater tuberosity of humerus incomplete, 94
ligaments. *See individual ligaments*
- Back, low sprain, 13-14
etiology 13 14
treatment, symptomatic 14
- strain, treatment, d-tubocurarine (Tubadil)
See d Tubocurarine (Tubadil)
use in acute back strain
- Baker cyst of diagnosis, differential from cyst of semilunar cartilages of knee 33
- Bandage, compression elastic postoperative care of knee in dislocation of patella, inadequacy of 3
"locked," 53
- sprain, ankle joint, 114-115
swellings about wrist 11
- gauze sprain, ankle joint, 114
- Bankhart operation, recurrent anterior dislocation of shoulder joint, 87
- Barton J R use of arthroplasty and osteotomy in treatment of knee 4
- Bechtol C O investigations, anomalies of bicipital groove 73 76
- Biceps brachii muscle groove anomalies, 74-77
angle, variations and incidences, 73 76
investigations, 73 76
degenerative processes, 74-77
formation of bony excrescences, 75 77
long head, tenosynovitis *See Tenosynovitis*, bicipital
ruptures, 97 98
tendon fraying shredding and hypertrophy 74 75 77
wearing away of intertubular portion of sulcus 76 77
- Bleeding, after knee injury treatment, compression bandage 22 23
in sprain knee 20
- Bone bank, infected specimens, analysis by cases 164-165
pattern 163 164
refrigerated, evolution of 163
- Bone (Continued)
bank (Continued)
source of supply 164
statistical data 165 169
analysis, of infected bone specimens 164-165
of infections, 167 169
of operative results, 166-167
failures, 166
bacteriologic study of bank bone used at operation, 165
results of preserved homogenous grafting, 168 169
type of operation 166
See also Bone grafting
chondroblastoma, benign diagnosis, differential from giant-cell tumor of bone 140-141
cyst, aneurysmal diagnosis differential, from giant-cell tumor of bone 138 139
unicameral, diagnosis, differential, from giant-cell tumor of bone 141
fibroma chondromyxoid, diagnosis, differential, from giant-cell tumor of bone, 139 140
nonossifying diagnosis differential, from giant-cell tumor of bone 139
grafting avulsion fractures of tibial mal leolus, 198
early clinical studies 163
technics autogenous vs frozen homogenous bone comparative study 163-169
of use of supply material 164-165
See also Bone bank
navicular fracture 10
tumor giant-cell *See Giant-cell tumor of bone*
- Buck, Gordon, use of arthroplasty and osteotomy in treatment of knee 4
- Buck, traction of "locked" knee 44
- Bursitis, obturator internus 103
- Bursitis, collateral ligament diagnosis differential from menisci, traumatic lesions, 43
- Calcaneonavicular ligament anatomy 110 196
internal, anatomy 111
- Calcaneum, 111
- Calcification, para-articular Pellegrini-Stieda. *See Pellegrini-Stieda para-articular calcification*
Campbell Willis, technics for correction of dislocation of patella recurrent 6
simple 62 63
- Capsular ligament, of ankle joint, an 111 112 193
- Cast, walking, sprain an

- Hamstring tendons avulsion from ischial tuberosity 66-68
 case report 66-68
 incidence 66
- Hauser operation for dislocation of patella, re-current, 5-6
 simple, 62, 63
- Heat therapy knee injuries, 24 25
 restoration of impaired circulation dislocation or sprain of hip joint, 104
 sprain, back, 14
- Heel wedge sprain, ankle 16-18
- Hemangioma, knee, diagnosis, differential from menisci traumatic lesions 43
- Hemarthrosis, aspiration in dislocation of patella simple, 61
- Hematoma, after knee injury treatment, compression bandage 22, 23
 in sprain knee 20
 subperiosteal with subsequent calcification from dislocation of hip, 106
 in wrist, incision for evacuation, contraindicated 11 12
- Hemorrhage, in joint injury 9
- Hip joint, anatomy 99 100
 blood supply 100
 development, 101 102
 dislocation, 102, 104-106 108
 anterior 104
 congenital, 101
 damage to soft tissues, 102
 with fracture acetabulum, and damage to head of femur 102
 mechanism 102 104
 from paralytic or infectious lesions, causing flexion-adduction contractures or spasm, 105
 posterior 102, 104
 treatment, 104-106 108
 of aseptic necrosis 105
 conservative 104-105
 factors, 104
 surgical 105 106
 cup arthroplasty of Smith Petersen, 106
- Injuries of ligamentous and associated structures, 99 108
 clinical aspects, 102 108
 dislocation See Hip joint, dislocation
 limp of unknown origin in children 102
 movements, 101
 nerves, 100 108
 redislocation, 106
 relations, 100-101
- Hitchcock, H H investigations, anomalies of bicipital groove 73 76
 bicipital tenosynovitis, 70
- Humerus, atrophy of greater tuberosity and new bone formation on shaft, 76
 giant-cell tumor of bone, 135
 tuberosity greater avulsion fracture incomplete 94
 fracture with rupture of rotator cuff after dislocation of shoulder 96-97
- Hyaluronidase, injection, effusion in knee in joints 25
 sprain ankle joint, 17 114 116
 swellings about wrist, 11
- Hydrocortisone therapy lesions, traumatic of menisci, 44 45
 sprain finger trigger 10
 wrist, administration, local injection 9 10
 oral 9 10
 synovitis, acute, in osteoarthritic knees 15
 tenosynovitis biceps brachii muscle 79
- Hydrops, intermittent, diagnosis, differential, from menisci, traumatic lesions, 43
- Iliacus muscle, 100
- Iliofemoral ligament, anatomy 99 101 103
 extension of hip joint, 101
 trauma, in dislocation of hip joint, 106
- Iliopsoas muscle, flexion of hip joint, 101
 medial rotation of hip joint, 101
- Interosseous ligament of ankle joint, anatomy 109 110 112
- Ischiofemoral ligament, adduction of hip joint, 101
 anatomy 99 100 103
 strain from wide adduction of hip joint, 102
 trauma, in dislocation of hip joint, 106
- Jewett nail, fractures, femur neck, 213
- Joints. See *Individual joints*
- Jones Sir Robert, technique in surgical treatment of "locked" knee 52, 53
- Keith, Sir Arthur Law of Ligament, 27
- Knee, arthritis traumatic, acute See Arthritis, traumatic, acute, knee
 arthrodesis step-cut fusion with medullary nailing. See Knee, step-cut fusion with medullary nailing
 cartilages, semilunar cysts, 29 37
 after-care 34
 diagnosis, importance 29
 etiology 30-31
 first instance reported by Ebner 29
 incidence and location 29 30
 pathology 30-32
 prognosis, 34
 report of 18 cases, 35 36
 statistical analysis, 36
 size, 31
 treatment, 33 34

curate support. The walking cast controls swelling by circumferential pressure and active motion. Procaine injection therapy of Leriche eliminates pain so that active motion continues to regulate the circulatory mechanism of the extremity and stasis does not develop. All three methods are then compatible in skilled hands, and the choice of the method to use depends upon the skill and the training of the operator.

There are minor variations in the use of procaine injection. The classical method is to aspirate the hematoma and to inject it with 5 to 10 cc of 1 per cent procaine. The simultaneous injection of 500 units of hyaluronidase is considered to increase control of swelling. Multiple injections may be needed if both the anterior and the posterior short hinges are torn.⁹ Multiple injections can be avoided by blocking the sural nerve three finger breadths above the tip of the lateral malleolus; anesthesia of the fifth toe is an indication of a successful block.³ All three methods—local procaine, hyaluronidase and

sural nerve block—can be combined in a shotgun treatment. Of course it is mandatory that the relief obtained by the procaine injection be adequate for the patient to put on his shoes and to walk freely. Failure of the patient to do so indicates an inadequate block. Return of the stiffness and the pain at any time in the following 72 hours may require a second procaine block.

Convalescent sprains should have the lateral border of the heel wedged 3/16 inch (Fig. 13). Shoe repair men call this an outer "Dutchman." This not only gives the patient relief from discomfort but also affords him a sense of security. Such a wedge should be worn at least 3 months to prevent recurrence of the trouble. Finally the patient should be instructed in heel-cord exercises for after all a short heel cord is why he sprained his ankle.

Applying the knowledge of ankle sprains herein discussed, the routine presented in the chart at the bottom of the following page is suggested as ideal in the handling of ankle injuries.^{9,10}



FIG. 14. Roentgenogram of an ankle showing extensive osteoporosis 4 months following a self-treated ankle sprain.

- knee (*Continued*)
 cartilages (*Continued*)
 semilunar (*Continued*)
 diagnosis, differential 33
 physical examination 33
 symptoms, 32 33
 tear 14
 Charcot, diagnosis differential from menisci,
 traumatic lesions, 43
 step-cut fusion with medullary fixation,
 145 147
 fat pads, hypertrophied or pedunculated
 diagnosis, differential, from me-
 nisci, traumatic lesions, 43
 foreign bodies, diagnosis differential from
 menisci, traumatic lesions, 43
 fracture
 diagnosis, differential from sprain,
 20-21
 hemangioma, diagnosis, differential from
 menisci, traumatic lesions, 43
 injuries, 13 15
 arthritis. *See* Arthritis, traumatic acute
 knee
 locked" knee. *See* Knee "locked"
 synovitis. *See* Knee synovitis
 tear. *See* Knee, tear
 ligaments, avulsion, diagnosis, differential
 from sprain, 21 22
 with dislocation of tibia, 25 27
 injuries, 20-27
 diagnosis, 20-22
 treatment, 20 22 27
 aspiration in effusion 25
 cold, 23
 compression, 22, 23
 diathermy contraindicated, 25
 elevation, 23
 heat, 24-25
 hyaluronidase, 25
 massage 25
 plaster cast 25
 procaine contraindicated, 23
 rest, 23
 surgical, 27
 taping, "Duke Simpson," 24 25
 sprain, diagnosis, 20-22
 differential, from avulsion 21 22
 from fracture 20-21
 examination, 20-22
 treatment, 20
 See also Individual ligaments
 "locked," 14-15
 definition, 50 52
 diagnosis, 15
 prognosis, 53
 treatment, 14 15
 conservative Buck's traction, 44
 manipulation, 44
 Liebolt technic 50-52
- Knee (*Continued*)
 "locked" (*Continued*)
 treatment (*Continued*)
 postoperative 53
 surgical 25 52 53
 arthrotomy for removal of medial
 meniscus, 52
 Sir Robert Jones technic 52, 53
 unlocking by patient 50
 nerves 100 108
 pouches, synovial diagnosis, differential
 from cysts of semilunar cartilages
 of knee 33
 septic and osteomyelitis of compound frac-
 ture of upper end of tibia, 145 146
 snapping diagnosis, differential from me-
 nisci, traumatic lesions, 43
 sprain(s) 14
 classification, 24
 step-cut fusion with medullary fixation
 145 151
 case reports, 145 150
 history of technic 145
 operative technic 146-151
 possible complications, 150-151
 fracture of nail at knee 151
 at site of insertion of nail, 150-151
 of tibia below pin, 151
 synovium diagnosis, differential, from me-
 nisci traumatic lesions, 43
 synovitis, acute in osteoarthritis, 15
 chronic 149
 tear cartilage 14
 ligament, lateral, internal, 13 15
 treatment 14
 tuberculous, step-cut fusion with medullary
 fixation, 148-150
 tumors, diagnosis, differential, from cysts of
 semilunar cartilages, 33
 xanthoma, diagnosis, differential, from me-
 nisci, traumatic lesions, 43
 Krognus operation for dislocation of patella,
 recurrent, 5
 simple 62 63
- Lateral ligament, external, of ankle joint,
 anatomy 110
 internal of ankle joint, anatomy 110 111
 of knee, tear 13-15
 treatment, 14
 Leriche method of procaine injection for
 sprained ankle 16, 17
 Ligament(s)
 acetabular transverse 99
 calcaneofibular anatomy 110 193
 calcaneonavicular anatomy 110 196
 internal, 111
 capsular of ankle joint, anatomy 99 111
 112, 193
 collateral. *See* Collateral ligaments

Ligament(s) (Continued)

- cruciate *See* Cruciate ligament(s)
- deltoid, of ankle anatomy 185 186 193 196 200 203
 - tear in fracture of fibular malleolus, 202
- iliofemoral anatomy 99 101 103
 - extension of hip joint, 101
 - trauma, in dislocation of hip joint, 106
- interosseous of ankle joint, anatomy 109 110 112
- ischiofemoral. *See* Ischiofemoral ligament
- knee *See* Knee, ligaments
- lateral. *See* Lateral ligament
- Law of Sir Arthur Keith 27
- medial of knee avulsion, 27
 - injuries, treatment, 27
- patellar transplantation for amputee dislocation of patella 62, 63
- pubofemoral, anatomy 99 100
 - strain, from wide abduction of hip joint, 102
 - trauma in dislocation of hip joint 106
- talocalcaneal, internal, anatomy 110
- interosseous, anatomy 110
- posterior anatomy 111 112
- talofibular anterior 110 185 186
 - posterior 185
- talonavicular anatomy 110-111
- tibiofibular antero-inferior anatomy 110
- posterior anatomy 110
- posteroinferior anatomy 112
- transverse of ankle joint anatomy 40 109-110
- See also individual names*
- Ligamentum teres, anatomy 110
 - trauma, in dislocation of hip joint, 106
- Limp of unknown origin, in children, 102
- Lipoma, knee joint, region diagnosis differential from cysts of semilunar cartilages 33
- Lippmann R. K. investigations and conclusions, bicipital tenosynovitis, 70
- Loose bodies, osteocartilaginous ankle joint, 206-207
- Lymph nodes cervical, tuberculous, early differential diagnosis from torticollis, 12
- Lymphatics, ankle joint, 183 185
- McLaren J. S. surgical technic for recurrent dislocation of patella, 4
- McLaughlin nail, fractures, femur neck, 213
- McLaughlin operation transacromial approach, partial rupture of rotator cuff of shoulder 94
- McMurray osteotomy fractures femur neck 213 214
- Malleolus, external anatomy 112
 - internal, anatomy 112
 - avulsion surgical treatment, 117 118
- Massage injuries, of knee, 25
 - restoration of impaired circulation, dislocation or sprain of hip joint, 104
- sprain, ankle, 16-18
 - back 14
- Mauch H. P. technic for recurrent dislocation of patella, 5
- Medial ligament of knee avulsion, 27
 - injuries, treatment, 27
- Medullary nailing, closed of Colles fracture, 152 162
 - anatomic results, 158
 - dynamic factors 158-160
 - history of technic, 152 153
 - indications, 153
 - reconstruction, 155 158
 - removing pin, 161 162
 - return to function, 161
 - stability of fixation, 158
 - technical errors and complications, 158-161
 - mechanical irritation at head of pin not inserted sufficiently 160
 - overcorrection in Cotton Loder position, 161
 - of distal fragment in ulna deviation, 158 160
 - silver fork deformity 161
 - time of operation, 154-155
- with step-cut fusion of knee, 145 151
- case reports, 145 150
- Charcot knee 145 147
- complications, possible 150-151
- fracture of nail at knee 151
 - at site of insertion of nail 150-151
 - of tibia below pin 151
- operative technic, 146-151
- septic knee and osteomyelitis of compound fracture of upper end of tibia, 145 146
- tuberculous knee 148-150
- Membrane synovial hip joint, 100
- Meniscectomy medial bucket handle tear 42 45
- Meniscus(1) cyst, 30 31
 - diagnosis, differential from parameniscal cyst, 32
 - injuries with collateral ligament sprain, 25
 - lateral, attachment, lesion of 40
 - lesions, traumatic incidence 39
 - lesions nontraumatic 43
 - rotation strain 43
 - tear or derangement, diagnosis, procaine infiltration 23

Meniscus(i) (Continued)**lesions (Continued)**

- traumatic, 39-46
 - anatomy 39 40
 - complications, 39 46
 - diagnosis 40-42
 - aspiration of effusion 41
 - differential 43
 - palpation 41
 - procaine injection 41
 - tests, 41-42
- pathology 39-40
 - bucket-handle tears, 39
- prognosis, 45-46
- symptomatology and physical findings, 40-42

treatment, conservative 43-44

- aspiration of joint, 44
- hydrocortisone instillation 44
- plaster cast, 44
- pressure dressing and ice bags, 44
- quadriceps exercises, 44
- postoperative 45
- surgical 44-45
 - approaches, 44
 - associated lesions, 45
 - hydrocortisone instillation into knee 45
 - incisions, 44
 - meniscectomy 42, 45

medial injuries, 48-53

- diagnosis, 49 50
 - history from patient 49
 - physical findings, 49
 - signs, 49 50
 - Leibolt, 49-50
 - McMurray 49 50
- incidence, 48
- "locking" of knee *See* Knee "locked"
- symptoms, 48-49
- traumatic, incidence, 39
- types, 48 49

Mercurochrome, painting of skin, sprain ankle joint, 113**Metacarpal, first, fracture 10****Meyer A. W., investigations, anomalies of bicapital groove, 76****Murphy sign of in diagnosis of fracture of navicular bone, 10****Muscle(s) adductor *See* Adductor muscle****genitellus(i) 101 107****gluteus. *See* Gluteus muscle****iliacus, 100****iliopsoas, flexion of hip joint 101****medial rotation of hip joint, 101****obturator *See* Obturator muscle****pectineus, 100****flexion of hip joint, 101****piriformis, anatomy 101 107****Muscle(s) (Continued)****popliteus tendon 40****psoas, 100****quadratus femoris 101 107****rectus femoris, anatomy 100 108****flexion of hip joint 101****sartorius, anatomy 108****flexion of hip joint, 101****Musculocutaneous nerve neurectomy 204****Musculotendinous cuff of shoulder *See* Rotator cuff of shoulder****Mutter pioneer work in surgical treatment of knee, 4****Myeloma. *See* Giant-cell tumor of bone****Myositis ossificans, with sprain ankle 18****Nail, Jewett, fractures, femur neck, 213****McLaughlin fractures, femur neck, 213****Smith Petersen, fractures, femur neck, 213****Navicular bone, fracture 10****Neck, inflammation deep-seated, differential diagnosis from torticollis, 12****sprain, 12 13****diagnosis, differential, 12****treatment, 12 13****ethyl chloride spray 12****Thomas collar 12****traction intermittent or continuous, 12 13****whiplash injuries, 12****wry *See* Torticollis****Necrosis, aseptic, in dislocation of hip traumatic 105 106****definition, 209****fractures, femur neck, 209****avascular in dislocations and fractures of talus, 185 190****attachments, 185 186****behavior of broken bones 186-190****extent of articular coating 185****fulcra and grooves, 185****growth pattern, 185****nutrient foraminae 186****proximity of vascular networks and laxity of intervening tissues, 186****Nerve musculocutaneous, neurectomy 204****obturator *See* Obturator nerve****to quadratus femoris muscle, 100 107****branches, trauma, in dislocation of hip 106 108****to rectus femoris muscle 100****saphenous, infrapatellar branch, 41 44****sciatic anatomy 100 107****trauma in posterior dislocation of hip joint, 105****sural, block, for sprain, ankle, 17****to vastus medialis muscle 108****Neurectomy musculotaneous nerve, 204**

- Nitrous oxide, with thiopental sodium, anesthesia for surgical treatment, menisci, traumatic lesions, 44
- Nutrient artery fibular shaft, 190
- Ober operation for simple dislocation of patella, 62 63
- Obturator artery 100
- Obturator muscle externus, anatomy 100 107
flexion of hip joint, 101
laceration in dislocation of hip joint, 106
internus, anatomy 101 103 107
trauma in dislocation of hip joint, 106
- Obturator nerve, 100
anterior anatomy 103
branches, trauma in dislocation of hip joint 106 108
articular branch, anatomy 103 107
posterior anatomy 103 107
- Osgood-Schlatter disease, diagnosis, differential from menisci, traumatic lesions, 43
high patella in boys after 56-57
- Osteochondritis, ankle joint, 205 207
dissectans, 205
diagnosis, differential from menisci, traumatic lesions, 43
tall, 205
- Osteochondromatosis, diagnosis, differential, from menisci, traumatic lesions, 43
- Osteoclastoma. *See* Giant-cell tumor of bone
- Osteomyelitis, of compound fracture of upper end of tibia with septic knee, 145 146
hemorrhagic, nonsuppurative, chronic. *See* Giant-cell tumor of bone
- Osteoporosis, from maltreatment of sprains of ankle joint, 119
with sprain, ankle, 17 18
- Osteotomy of femur lower end, with correction of genu valgum, for recurrent dislocation of patella, 58
in knee operations, use by early American surgeons, 4
valgus of knee 62 64
- Paralysis, ischemic, Volkmann's, with sprain, ankle, 18
muscles, below knee from trauma to sciatic nerve in dislocation of hip joint, 105
thigh, posterior from trauma to sciatic nerve in dislocation of hip joint, 105
- Pasteur F investigations and conclusions, bicipital tenosynovitis, 69 70
- Patella, chondromalacia, diagnosis, differential, from menisci, traumatic lesions, 43
- Patella (*Continued*)
dislocation complications, 61
congenital. *See* Patella, dislocation, recurrent
diagnosis differential from menisci, traumatic lesions 43
etiology 61
mechanism, 61
recurrent, 55-60
definition, 55
etiology 55-56
anatomic developmental abnormalities, 56
high position after Osgood-Schlatter disease 56-57
vastus internus, undeveloped, 56
postoperative care, 6
surgical treatment, 3-7 57-60
Campbell technique, 6
complications, 6
by early American surgeons, 3-4
Goldthwait technique, 5
Hauser technique, 5-6
incision on outer side and "reefing" on inner 4
"knee cage" brace of Pearson, 4
Krogius technique 5
McLaren technique, 4
Mauck technique 5
osteotomy of lower end of femur with correction of genu valgum 58
patellectomy 6-7
condemned 59-60
patelloplasty after patellectomy 7
Perkins technique, 4
pin fixation through femur and tibia, 6
Pollard technique 4
requirements, 3
Soutter technique, 5
Steindler technique 6
transposition, of insertion of patellar tendon, 58
of tibial tubercle, 58-59
simple, treatment, conservative 61-62
aspiration of hemarthrosis or effusion 61
immobilization 61
surgical, 62-65
Albee technique, 62, 64
Campbell technique, 62, 63
criteria, 62
Galle technique, 62, 63
Goldthwait, 62, 63
Hauser technique, 62, 63
Krogius technique, 62, 63
Ober technique 62, 63
Teal technique 62, 64 65

Patella (Continued)

- fracture, surgical treatment
 - pins, Anderson technic 4
 - high position in chondromalacia 57
- after Osgood Schlatter disease 56-57
- loose bodies, diagnosis, differential from
 - meniscus, traumatic lesions, 43
- locking. See Patella, dislocation
- recurrent
 - luxation, with spontaneous return to normal position 55
- ligament, transplantation for simple dis-
 - location of patella, 62 63
- ectomy for recurrent dislocation 7
- condemned as treatment, 59-60
- plasty after patellectomy 7
- use of "knee cage" brace to stabilize
 - slipping patella, 4
 - muscle, 100
- location of hip joint, 101
- Pellegrini Stieda disease. See Pellegrini Stieda
 - para articular calcification
- Pellegrini Stieda para articular calcification
 - 121 126
 - clinical picture, 125 126
 - etiology 121 122
 - history 121
 - pathogenesis, theories, 125
 - pathology 122 125
 - treatment, 126
- Penicillin therapy menisci traumatic lesions,
 - postoperative parenteral adminis- tration, 45
- Pentothal Sodium. See Thiopental sodium
- Periosteum damage, from dislocation of hip joint, 106
- Perkins, J W surgical technic for recurrent
 - dislocation of patella 4
- Peroneal artery 180 183
- Physiotherapy restoration of impaired circula-
 - tion, dislocation or sprain of hip joint, 104
- Piriformis muscle, anatomy 101 107
- Plantar artery lateral 180
- Plaster cast, contraindicated, after reduction of
 - "locked" knee 53
- dislocation of patella, simple, 61-62
- fracture and dislocation of hip joint, 104
- redislocation of hip joint, 106
- with walking heel sprain, ankle joint, 115
- Pollard, surgical technic for recurrent disloca-
 - tion of patella, 4
- Popliteal artery anatomy 107 180
- injury 25 26
- Popliteus muscle tendon, anatomy, 40
- Pouch, suprapatellar closed, with chondro-
 - malacia of patella, 57
- Procaïne block lumbar sympathetic ganglia
 - for Sudeck's atrophy 120

Procaine (Continued)

- injection
 - in diagnosis, rupture of rotator cuff of shoulder without dislocation or fracture 93 94
 - traumatic lesions of meniscus 23 41 44
 - injuries of knee ligaments, contraindicated, 23
- for sprains, ankle joint, 113
- Leriche technic, 16 17
- back, 14
- Psoas muscle 100
- Pubofemoral ligament, anatomy 99 100
- strain from wide abduction of hip joint, 102
- trauma, in dislocation of hip joint, 106
- Pudendal artery internal, capsular branches 100
- trauma in dislocation of hip joint, 106
- Putti Platt operation recurrent anterior disloca-
 - tion of shoulder joint, 87
- Quadratus femoris muscle 101 107
- Radius, giant-cell tumor of bone 135
- head, fracture effect on wrist motion, 10-11
- relationship to ulna, 136
- Rectus femoris muscle anatomy 100 108
- flexion of hip joint, 101
- Rest as therapy knee injuries, 23
- Rete tarsal, lateral, 180-181 183 193
- tibial malleolar 181 183 193
- Retinacula, hip joint, 99 100
- Röntgen ray therapy deep from Sudeck's
 - atrophy 120
- Rotation, hip joint, lateral, 101
- medial, 101
- Rotator cuff of shoulder anatomy 92
- degenerative lesions, 74 77
- rupture(s) 92-98
 - after dislocation of shoulder 96
 - and fracture of greater tuberosity 96-97
 - without dislocation of fracture 92 96
 - diagnosis, arthrography 93 94
 - "drop-arm sign," 93
 - procaine injection, 93-94
 - site 92 93
 - types, 93
- massive complete separation of cuff 95
- surgical treatment, 95 96
- partial confirmation at operating table by
 - buckling of tendon with instrument, 95
- locating lesions at operating table 95
- treatment, conservative 94
- surgical, 94-95
- Saphenous nerve infrapatellar branch, 41 44
- Saphenous vein, long, 183 184
- short, 183

- Sarcoma, myeloid. *See* Giant-cell tumor of bone
- Sartorius muscle anatomy 108
flexion of hip joint 101
- Saunders investigations and conclusions, bicapital tenosynovitis, 70
- Schrager V. L., investigations and conclusions bicapital tenosynovitis, 70
- Sciatic nerve anatomy 100 107
trauma in posterior dislocation of hip joint, 105
- Sex as factor incidence, giant-cell tumor of bone, 134
- Shoulder joint, acromioclavicular joint. *See* Acromioclavicular joint
- dislocation recurrent anterior 86-90
analysis of results, 90
incidence, 86
pathologic anatomy 86-87
postoperative treatment, 90
surgical treatment, 87 90
Magnuson technic (modified) 88 90
types of arrangement of synovial recesses 86 87
rupture of rotator cuff after 96
with fracture of greater tuberosity 96-97
- frozen degenerative and inflammatory changes in soft tissue components of scapulohumeral articulation 71 72
- pain and stiffness from bicapital tenosynovitis, 69
- rotator cuff *See* Rotator cuff of shoulder
- sprain, 10-12
- Skin, graft, after neurectomy of musculocutaneous nerve 204
- necrosis, in dislocations and fractures of talus *See* Necrosis, avascular in dislocations and fractures of talus
- slough compound fracture-dislocation of talus 201
- ulcers, from crushing ankle injuries, 200 202
- Smith S. Alwyn, technic for reconstruction of cruciate ligaments of knee 5
- Smith Petersen cup arthroplasty for dislocation of hip with aseptic necrosis of head of femur 106
- incision dislocation of hip joint, anterior 106
- nail, with plate fractures, femur neck, 213
- Soutter technic for recurrent dislocation of patella, 5
- Spine, tuberculosis, early diagnosis, differential, from torticollis, 12
- Sprain(s) 8 19
ankle *See* Ankle, sprain
articular 9
back, low *See* Back low sprain
claims against insurance companies, 8
- Sprain(s) (Continued)
classification 9
definition, 8-9
finger 9
incidence, Blue Shield statistics, 8
knee, 14
classification 24
ligaments. *See* Individual ligaments
muscular 9
neck. *See* Neck, sprain
shoulder 10-12
thumb 10
wrist. *See* Wrist, sprain
washerwoman's, 9
- Steindler A., technic for recurrent dislocation of patella, 6
- Stocking, elastic, for postoperative protection and support of knee in dislocation of patella, inadequacy of 3
- Strain, as medical term, 8
- Strapping with adhesive tape, sprain, ankle joint, 113 114
- Sudeck, atrophy of. *See* Atrophy Sudeck's
- Supraspinatus tendon rupture, 92
partial, 94
- Sural nerve, block, for sprain, ankle, 17
- Sustentaculum tali, 111
- Swelling, in joint injury from hemorrhage 9
- Sympathectomy periarterial, for Sudeck's atrophy 120
- Synovialus, plica, extensive, with chondroma lacia of patella, 57
- Synovialoma knee, diagnosis, differential from menisci, traumatic lesions, 43
- Synovitis, acute, in knees, osteoarthritis, 15
chronic, of knee, 149
diagnosis, differential, from menisci, traumatic lesions, 43
- Talocalcaneal ligament internal anatomy 110
interosseous, anatomy 110
posterior anatomy 111 112
- Talofibular ligament, anterior anatomy 110 185 186
posterior anatomy 185
- Talonavicular ligament, anatomy 110 111
- Talus, dislocation, with avascular necrosis, 189
without avascular necrosis, 188
foramenae nutrient, 186, 193
fracture compound, and dislocation, reduction, 190
skin slough 201
and dislocations, avascular necrosis. *See* Necrosis, avascular in dislocations and fractures of talus
transcervical with dislocation partial, reduction 188 190
without dislocation, conservative treatment, 188

Talus (Continued)

- inclination and alignment during growth 187
- ossification, 186
- osteochondritis, 205
- structure and mechanism, 109 185 187
- Taping, "Duke Simpson" knee injuries, 24 25
- as treatment in sprain, ankle, condemned 16
- Tarsy J. M., investigations and conclusions, bicipital tenosynovitis 70
- Teal operation for dislocation of patella re
 - current, 6-7
 - simple, 62, 64 65
- Tendon(s) ankle, 192 193
 - biceps brachii. *See* Biceps brachii tendon
 - hamstring, avulsion from ischial tuberosity 66-68
 - case report, 66-68
 - incidence, 66
 - obturator internus muscle interposition between neck of femur and acetabulum in dislocation of hip joint, 106
 - patellar transposition, for recurrent dislocation of patella, 58
- "Teno-burite," term of Pasteur for new syndrome 69
- Tenosynovitis, bicipital 69-85
 - analysis of cases reviewed, 81-85
 - poor results, 84-85
 - clinical features, 77 79
 - after age of 30 years, 78-79
 - in young, 77 78
 - etiology 73-74
 - anatomic and functional factors, 73-74
 - incidence, 70
 - pathology 70-73
 - postoperative management, 81
 - shoulder joint pain and stiffness from, 69
 - investigations and conclusions, 69 70
 - treatment, conservative 79
 - surgical, 79-81
 - stenosing, chronic, 9
- Thigh, posterior muscles, paralysis, from trauma to sciatic nerve in hip dislocation, 105
- Thiopental sodium with nitrous oxide anesthesia for surgical treatment, menisci, traumatic lesions, 44
- Thomas collar for support in neck sprain 12
- Thumb extensor tendon sheath, constriction
 - chronic 9
 - sprain, 10
- Tibia, dislocation, from avulsion of knee ligaments, 25-27
 - fracture, below pin in medullary nailing with step-cut knee fusion, 151
 - comminuted, of bearing plate, treatment, 193
 - with traumatic lesions of menisci, 39

Tibia (Continued)

- giant-cell tumor of bone 135 136
- malleolus, anatomy and development, 195 196
 - fracture, avulsion 194-196
 - nonunion 196
 - dowel graft, 196 198 199
 - treatment, surgical, 196 198 199
 - vascular damage 198
 - vascular relations, 197
 - compression 194
 - vascular relations, 197
- spurs from articular surfaces, diagnosis, differential, from menisci, traumatic lesions, 43
- synovia or articular surfaces, loose bodies, diagnosis, differential from menisci, traumatic lesions, 43
- tubercle transposition for recurrent dislocation of patella, 58
- Tibial artery anterior 180
 - displacement, 191
 - posterior 180
 - tear 191
- Tibiofibular ligament, antero-inferior anatomy 110
 - posterior anatomy 110
 - postero-inferior anatomy 112
- Toe, sprain, treatment, 9
- Torticollis, 12
- Traction, Buck's, "locked" knee 44
 - dislocation, hip joint, 105
- Transverse ligament of ankle joint, anatomy 40 109 110
- Tubadil *See* d Tubocurarine
- d Tubocurarine (Tubadil) introduction into modern medicine and experimental use, 172
 - use in acute back strain, 172 175
 - case reports, 173 175
 - contraindications, 173
 - dosage, 172
 - overdosage 173
 - side reactions, neostigmine as corrective, 173
- Tumeur à myélopaxes *See* Giant-cell tumor of bone
- Tumor(s) giant-cell benign *See* Giant-cell tumor of bone
 - of bone. *See* Giant-cell tumor of bone
 - knee. *See* Knee tumors
- Ulcers, skin from crushing ankle injuries, 200 202
- Ulna, giant-cell tumor of bone, 135
 - relationship to radius, 156

Vasospasm segmental after panstragalar
arthrodesis, 204

Vastus muscle lateralis anatomy 108
medialis, anatomy 108

Vein(s) ankle joint, 183 185
foot, direction of flow 183
saphenous long, 183 184
short 183

Wrist, fracture, navicular bone, 10

sprain, fracture of navicular 10

swelling, treatment, 11

treatment, hydrocortisone, 9 10

Xanthoma, knee diagnosis, differential from
menisci, traumatic lesions, 43

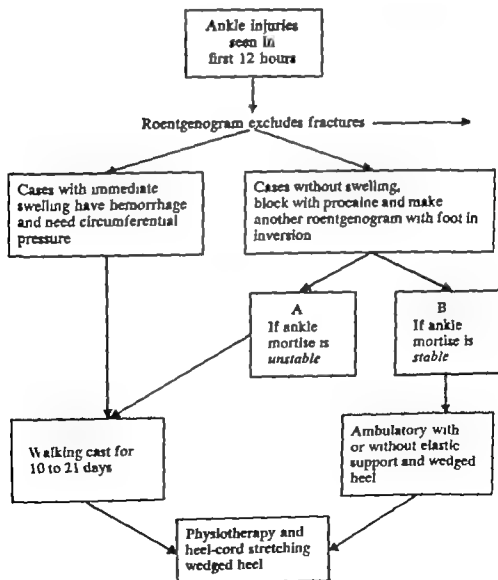
Zona orbicularis, anatomy 99

Failure to treat joint injuries properly is to invite some rather serious complications such as

- 1 Reflex dystrophy osteoporosis and allied conditions (Fig 14)
- 2 Volkmann's ischemic paralysis
- 3 Gangrene
- 4 Traumatic arthritis
- 5 Myositis ossificans

The greatest single error in treatment is failure to handle the local situation adequately so that the injured part has the necessary amount of support, plus a reasonable amount of active function. Fingers are made to be used and feet are made to be walked upon neither function well in bed. Pain and secondary swelling in untreated cases which

will keep a hand immobilized until the fingers are stiff must be eliminated. On the other hand, inexpertly applied, bulky or inadequate supporting bandages, splints or casts are one of the most vicious factors in producing complications far worse than the original injury if left untreated. Forceful manipulation of a painful joint by a therapist will do further irreparable damage. The safe axiom is that vigorous massage and forcible passive motion are extremely detrimental in all fresh and most old injuries. The Rule exercises must never cause pain. It also follows that injuries which result in joint instability if left untreated will gradually wear out that joint as surely as a misaligned axle does a bearing.



CONCLUSION

A sprained ankle is the most common athletic injury. Unfortunately, often these conditions are considered as minor and are self-treated. Proper treatment can reduce materially the period of temporary disability and prevent more serious complications, which cause permanent partial disabilities.

REFERENCES

- 1 Babcock, W. Wayne. *Principles and Practice of Surgery* p 759 Philadelphia: Lea & Febiger 1944.
- 2 Collins, Thomas. Personal communication.
- 3 Gay, J. R. and Abbott, K. H. Common whiplash injuries of the neck. *J.A.M.A.* 52, 1698 1953.
- 4 Hughes, J. Rowland. Sprains and subluxations of the ankle joint, *Proc Roy Soc Med* 35 765 1942.
- 5 Hutchison, James B. Sural nerve block for ankle sprains. *U.S.A.F. Med. J.* 2:799 1951.
- 6 Leonard, Morton H. Injuries of the lateral ligaments of the ankle. *J. Bone & Joint Surg.* 31A 373 1949.
- 6a Leriche, R. *The Surgery of Pain* p 16. Translated by A. Young, Baltimore: Williams & Wilkins, 1939.
- 7 MacAusland, W. R., Gartland, John J., Hallock, Halford. The use of hyaluronidase in clinical orthopedics. *Scientific Program Am. Acad. Orth. Surg.* Jan. 1953. to be published at a later date.
- 8 Murphy, John B. Knuckle percussion test. *Clinics of John B. Murphy* No. 3 1390 1915.
- 9 Pipkin, Garrett. Sprains, *J. Missouri M. A.* 45:569 1948.
- 10 ———. Prognosis on sprains, *J. Insur. Med.* 4 17 1949.
- 11 Schlemmer, Lee C. *Scientific Program, Am. Acad. Orth. Surg.* Jan. 1953. Discussion of reference 7 as above.

linitive method of distinguishing between fracture and sprain. In addition to the usual anteroposterior and lateral views, the so-called tunnel view taken with the knee flexed to show the intercondylar notch in sharp relief always should be included. Also when tenderness is marked at the tibial plateau oblique views frequently will disclose frac-

ture lines not visible in other projections. When the injured knee is seen within moments of the accident, usually it is possible to sort out complete avulsions from less severe sprains. In a series now totaling 12 complete avulsions of collateral ligaments, 2 simple clinical signs were found to be of great help.² The first of these is the presence

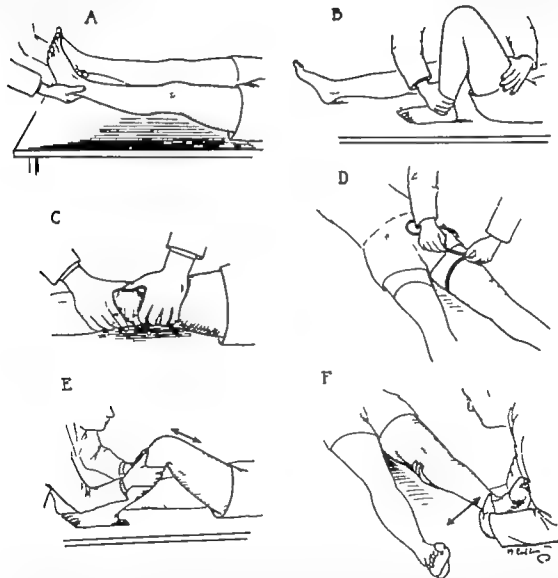


FIG 1 Basic steps in examination of the knee. The subject is supine and completely relaxed. All steps are first carried out on the uninjured leg. (A) Inspection and determination of extension. (B) Determination of passive flexion. This follows a comparison of active flexion with the uninjured leg and is not carried beyond the point of pain. (C) Palpation for fluid. Pressure on the suprapatellar pouch drives small quantities of fluid downward. The right index finger is shown about to compress the anterolateral aspect of the joint, producing a fluid wave palpable by the thumbs and the left hand. (D) Determination of muscle atrophy. The circumference of the thighs is measured at the same level. (E) Determination of cruciate ligament laxity. The knee is flexed at 90°. The examiner's forearm presses firmly against the lower tibia while both hands grasp the leg just below the knee and manipulate it in the sagittal plane. (F) Determination of collateral ligament laxity. One of the examiner's hands rests on the table and firmly grasps the femoral condyles. The other grasps the ankle and manipulates the extended knee in the coronal plane. (Quigley T. B. Surg., Gynec. & Obst. 87:569)

of sufficient relaxation to permit 15° or more of abduction or adduction of the fully extended tibia on the femur as compared with the opposite uninjured knee. This sign is based on the clinical and experimental work of Palmer,⁴ Branagan and Voshell,⁵ and Abbott and his co-workers.⁶ It can be considered as absolute evidence of complete avulsion of the involved collateral ligament, and either avulsion or severe stretching of the anterior cruciate ligament. The other sign, which is present less often but constitutes valuable evidence when it is encountered, consists of a diffuse doughy subcutaneous edema of the anterior, the medial and lateral aspects of the joint. This is seen only occasionally immediately after injury and is encountered much more often hours or days later. It is caused by rupture of the joint capsule accom-

panying the ligament avulsion. Fluid forming in the joint in response to the injury escapes through this rent and disseminates in the same manner as a subcutaneous hypodermoclysis. When either or both of these signs are encountered and preliminary roentgenograms have shown no evidence of bone injury, prompt examination under anesthesia is indicated. If manipulation in abduction or adduction under anesthesia confirms the pre-anesthetic impression, suitable exposure and prompt surgical repair are indicated.

The diagnosis of less severe sprains cannot be established accurately on the day of injury. It is sufficient on that day to rule out fracture, complete ligament avulsion, or internal derangement of such degree as firmly to lock the joint, and to determine the side of the joint involved in the sprain. Four car-

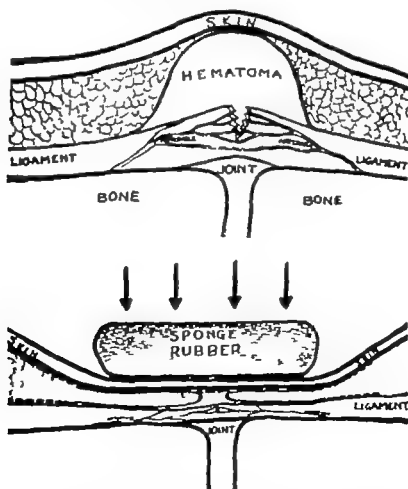


FIG. 2 The effect of compression on a typical ligament sprain.
(Quigley T. B. Mod. Med. 20:150)

dinal principles of immediate treatment based on the natural history of the ligament injury can be implemented. These are

1 *Cold* to constrict the arteriolar bed and diminish hematoma formation

2 *Compression* also to check active bleeding in the injured tissues and to disseminate hematoma already formed. The absorption of a hematoma is a function of its surface area. If a single hematoma is disseminated through tissue planes through compression and converted into X hematomata the surface area of the mass will be increased by more than λ^2 . Also the resulting scar will be thin and pliable rather than massive (Fig 2)

3 *Elevation* of the injured part will decrease edema formation by simple hydrostatics

4 *Rest* either in bed or on crutches. It is obvious that the more a recent injury is aggravated the greater will be disability both in time and degree. After a few hours cold is of little value, but compression rest and elevation are useful for several days. Procaine has no place in the treatment of knee ligament injuries. It does not promote the natural process of healing, and with pain abolished a minor sprain can easily become major. Rarely the pain imposed by a medial ligament sprain will fix the joint so firmly in flexion as to raise the question of concomitant mechanical block from a torn and deranged meniscus. The diagnosis can be settled by procaine infiltration of the injured ligament. Both cold and compression must be used with care if at all in the aged and in patients with peripheral vascular disease of any description. Foam rubber cut to a key hole shape is an effective means of achieving compression (Fig. 3). Fluid in the knee can distend the joint only anteriorly, therefore compression need be exerted only against the suprapatellar pouch and along the sides of the patella and the infrapatellar tendon. One layer of sheet wadding is placed between the skin and the rubber since a few patients are sensitive to chemicals in the rubber. If foam

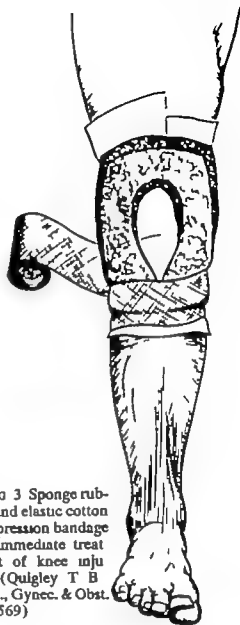


FIG 3 Sponge rubber and elastic cotton compression bandage for immediate treatment of knee injuries (Quigley T B Surg., Gynec. & Obst. 87 569)

rubber 1 inch thick of the "tan-open-cell" type is compressed to one half its thickness a force between 40 and 60 Gm per cm is exerted. Empirically this has been found to be sufficient to increase the intra-articular pressure to a point above the filtration pressure of the synovia. If such a compression dressing is applied before effusion has occurred and is kept in place for several days effusion usually does not occur. From 24 to 48 hours after the injury and after institution of immediate therapy an accurate determination of the ligament involved, together with the degree of its involvement, can be made. Tenderness now is localized and can be elicited either by direct pressure or by

manipulation producing stress on the ligament. With a little experience sprains can thus be classified as Grades I to IV. Grade I is the least degree of sprain that can conscientiously be called a sprain. Grade IV is complete avulsion of the ligament. Such classification is useful in prognosis. For example, it has been well established that a college varsity football player sustaining a

Grade II sprain of the tibial attachment of the medial collateral ligament will be sufficiently recovered to resume football 3 weeks after injury provided that immediate and subsequent treatment has been carried out carefully.

Also from 24 to 48 hours after injury heat rather than cold can be employed, since the danger of further bleeding is past. Any sort

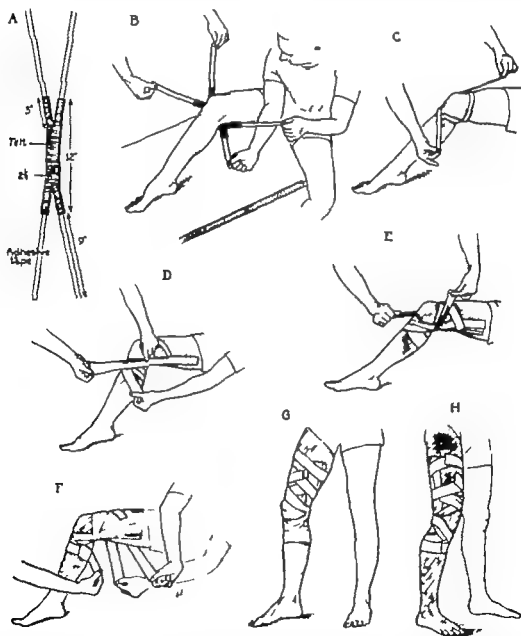


FIG. 4 The "Duke Simpson" protective knee taping. (A) Details of the cruciate-ligament supporting component. (B and C) Application of the cruciate-ligament supporting component. (D) Application of medial and lateral collateral ligament supporting components, 3 on each side. (E) Reinforcement of cruciate-ligament supporting component. Two strips of tape are applied crossing anteriorly above and below the joint. (F) Application of circular anchoring tapes. These are snug but not constricting. (G and H) The complete taping. (Quigley T. B. Surg. Gynec. & Obst. 87: 569.)

of wet heat is satisfactory, diathermy is contraindicated. Recently injured joints treated by diathermy almost invariably develop further effusion. Exercise up to but not beyond the point of pain is instituted, first in the form of quadriceps setting and as soon as possible as progressive resistance exercise.⁸ Gentle centripetal massage is of great value if done skillfully in relaxing the spasm of apprehension and initiating active motion. Clumsy and heavy-handed massage is worse than useless. If effusion occurs to the point of lifting the patella aspiration should be carried out. This is not a casual office procedure. The antibiotics have not decreased the sensitiveness of the knee to infection. An aspiration is really the same as an arthrotomy and the same safeguards and precautions should be observed. The needle should be of large caliber (8 or 10) and should be introduced from the side at the level of the patella into the suprapatellar pouch *never* through the quadriceps. The author's experience with hyaluronidase¹⁰ in facilitating the resorption of bloody effusion is too limited to justify critical comment. However it seems to be a safe and reasonable adjuvant to treatment but not a substitute for aspiration.

Immobilization in plaster has little place in treatment in most ligament injuries. Atrophy of the quadriceps particularly of the vastus medialis occurs very rapidly and can be overcome only by prolonged and tedious progressive resistance exercise. Atrophy is much better avoided than overcome. Weight bearing should be permitted only when normal walking can be accomplished without pain or limp. In athletes it must be remembered that the sprained ligament heals with scar and scar is always less resilient than the tissue it replaces. Accordingly protection must be given the injured ligament when the athlete resumes activity. For more than 50 years various methods of protection of the injured knee have been subjected to critical appraisal in the Medical Department of the Harvard Athletic Association. Various knee cages have been tried at various times but all share the underlying defect of being a

simple hinge. Unfortunately, the knee is far from a simple hinge. A toggle joint in the steels on the medial and the lateral aspects of a plastic or leather knee cage would approximate the natural motion of the knee but no toggle joint has yet been devised which can stand the stresses of contact sport. Adhesive taping has been very satisfactory. The "Duke Simpson taping" (Fig. 4) provides, in effect, an extra set of collateral and cruciate ligaments outside the skin which give effective protection for a full afternoon of vigorous sport. Eventually, the scar in the sprained ligament becomes replaced with elastic collagen fibers and becomes normal ligament. It is not known how long this process takes and at Harvard the rule has long been established that once a Grade II, III or IV sprain is sustained the player wears tape at every practice session or game in every contact sport for the rest of his college career. There is no doubt whatever that this program although at times tedious to the player and certainly expensive in terms of man hours of trainer's time has almost completely eliminated recurrence of knee ligament injury.

It is reasonable to assume that some degree of injury to the meniscus or to its peripheral attachments occurs with every collateral ligament sprain of the knee. Only a very few of these are so severe at the time of injury as to present the classic appearance of a locked knee requiring prompt surgery. The great majority come into clinical focus only after the ligament injury has healed and the symptoms of "giving way," recurrent locking, and the signs of recurrent effusion, tenderness and atrophy appear. In general, the diagnosis of internal derangement is one which grows on both the patient and the surgeon as such cases are followed. Operation should be carried out when the disability has reached the point of becoming a nuisance incompatible with the patient's normal activities and the surgeon is convinced that the knee is wearing out faster than the patient.

Fortunately avulsion of enough ligaments to permit complete dislocation of the tibia on the femur is rare. The possibility of injury

to the popliteal artery demands *immediate* reduction without waiting for roentgenograms or even anesthesia. If restoration of arterial supply does not follow reduction exploration of the popliteal space inspection of the damaged segment of artery and if necessary excision and vein or artery graft should be carried out at once followed by lumbar sympathectomy and heparinization. The ex-

perience of World Wars I and II with regard to injuries to popliteal arteries is discouraging,^{11,12,13} and only by such vigorous measures can gangrene be averted. When arterial supply is intact after reduction early exploration and repair of damaged ligaments is the treatment of choice.

The management of complete avulsion of collateral ligaments is still not established,

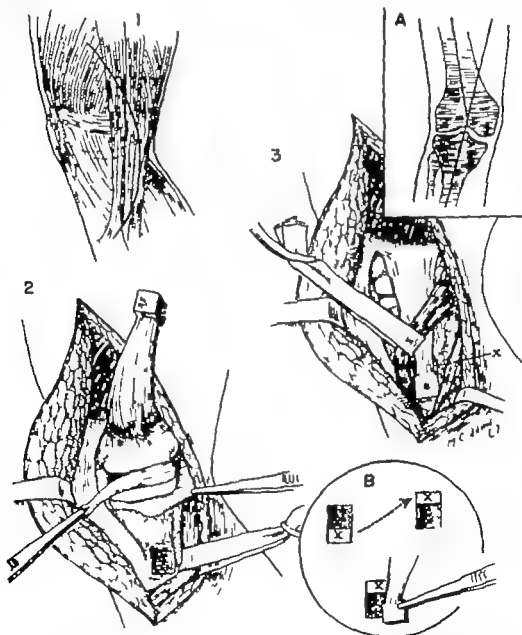


FIG. 5 Reconstruction of old avulsion of medial collateral ligament after the manner of Mauck. (A) Diagram of roentgen appearance of abducted fully extended knee under anesthesia. (1) The incision. A flap of skin and subcutaneous tissue is turned up. (2) The medial ligament, together with a block of bone at its tibial insertion, turned proximally. The gracilis is retracted. The meniscus is being removed. (3) The block of bone is replaced as far distally as necessary and secured with a screw. (B) Diagram showing the final position of the bone block and ligament reconstruction.

although considerable experience has accumulated in the literature in the past two decades in favor of prompt surgical exploration and repair. In no case in the author's experience has exploration of a joint presenting characteristic signs of complete ligament avulsion been found to be unnecessary, and in no case has the eventual disability after operation been anything but trivial. The operative approach in recent injuries consists of a curved incision, convex posteriorly and the upward dissection of a flap of skin and subcutaneous tissue. The whole anterior and medial (or lateral) aspect of the joint is thus exposed to view. Invariably the damage revealed is more severe than the surgeon anticipates. At times the avulsed ligament has been sucked into the joint itself. Almost always the meniscus is displaced or torn and requires removal. The author's policy with regard to the anterior cruciate ligament has been that of Darrach,¹⁸ who believed that formal restoration of this ligament was not necessary. The knee can be considered as a physiologic unit. The loss of one of its components is not serious and if this component is the anterior cruciate ligament it is even insignificant, provided that the musculature of the knee is normal. This is merely a restatement of Sir Arthur Keith's Law of Ligament.¹⁴ The ruptured anterior cruciate ligament is either reapproximated or if this is inconvenient, completely excised. The object of either procedure is simply to remove dangling ends which may become a source of future internal derangement. If the avulsion includes a portion of a tibial spine, reapproximation can be accomplished by a long mattress suture carried through drill holes in the tibia, from its anteromedial surface to the fracture face at the base of the tibial spine as described by O'Donoghue.¹⁵ Most avulsions of the medial and the collateral ligaments occur at their distal ends and present little difficulty in technical reconstitution. A few avulse at the femoral attachment but these also are not difficult to reattach. Fortunately a very few rupture at their mid portions for these a fascial graft or trans-

plantation of the sartorius tendon¹⁶ may be necessary.

Patients presenting themselves months after severe collateral ligament injuries will often describe instability and unsteadiness particularly in twisting or running motions and a sense of "giving way" which is more severe than the "giving way" associated with the damaged meniscus. The demonstration of laxity in the ligament may be difficult because of firm musculature which snaps into contraction at the first attempt to manipulate the extended joint. In these manipulations under anesthesia will solve the problem. If gross laxity of the medial ligament is encountered, exposure of the ligament and reattachment of its tibial end at a more distal point after the manner of Mauck¹⁷ is very satisfactory (Fig. 5). Chronic relaxation of the lateral collateral ligament is rare and is handled in the same general fashion.

SUMMARY

- 1 The natural history and the diagnosis of sprains and avulsions of ligaments of the knee are discussed.
- 2 A plan of treatment based on the pathology of the injury is presented.
- 3 The indications for and technical aspects of operative intervention are described.

REFERENCES

- 1 Miltner L. J. Hu C. H., and Fang, H. C. The pathology and treatment of joint sprain, *Chinese M. J.* 49:521 1935.
- 2 Ropes, M. W. and Bauer W. *Synovial Fluid Changes in Joint Disease* Cambridge Mass., Harvard, 1953.
- 3 Quigley T. B. The treatment of avulsion of the collateral ligaments of the knee, *Am. J. Surg.* 78:574 1949.
- 4 Palmer I. On the injuries to the ligaments of the knee joint, *Acta. chir. Scandinav.* 81:53 1938.
- 5 Brantigan, O. C., and Voshell A. F. The mechanics of the ligaments and menisci of the knee joint, *J. Bone & Joint Surg.* 23:44 1941.
- 6 Abbott, L. C., Saunders, J. B. deC., Bost, F. C., and Anderson, C. E. Injuries to the ligaments of the knee joint, *J. Bone & Joint Surg.* 26:503 1944.

- 7 Quigley T B The management of knee injuries incurred in college football, *Surg Gynec & Obst.* 87:569 1948
- 8 DeLorme, T L, and Watkins, A. *Progressive Resistance Exercise* New York, Appleton 1951
- 9 Thorndike, A. *Athletic Injuries* Philadelphia, Lea & Febiger 1948
- 10 MacAusland, W R., Jr., Gartland, J J and Hallock, H. The use of hyaluronidase in orthopedic surgery *J Bone & Joint Surg.* 35A:604 1953
- 11 Makins, G H. *On Gunshot Wounds to Blood Vessels*, Bristol, Wright, 1919
- 12 DeBakey M E. and Simeone, F A. Battle injuries of arteries in World War II analysis of 2471 cases, *Ann. Surg.* 123:534 1946
- 13 Warren, R. War wounds of arteries, *Arch. Surg.* 53:86 1946
- 14 Keith, Sir Arthur. *Menders of the Maimed* (Limited Edition) pp 76-77 Philadelphia, Lippincott, 1952.
- 15 O'Donoghue D H. Surgical treatment of fresh injuries to the major ligaments of the knee *J Bone & Joint Surg.* 32A:721 1950
- 16 McMurray T P. The operative treatment of ruptured internal lateral ligament of the knee *Brit. J Surg.* 8:377 1919
- 17 Mauck H P. A new operative procedure for instability of the knee *J Bone & Joint Surg.* 18:984 1936
- 18 Darrach, William. Internal derangements of the knee, *Ann. Surg.* 102:129 1935

Cysts of the Semilunar Cartilages of the Knee

LOUIS W. BRECK, M.D.*

INTRODUCTION

A very interesting lesion, cysts of the menisci was regarded in the past as very rare and since it is a benign lesion it was considered as being of little importance. However in the last 15 years a considerable number of good papers have been written on this subject, and it has turned out that the lesion is much more common than was supposed previously. Since it is associated so commonly with trauma, industrial surgeons and military surgeons have learned to be on the lookout for this lesion. In World War II there was a large amount of routine military training engaged in by soldiers whose knees were constantly subjected to trauma. This produced a relatively high incidence of knee cartilage cysts which in turn was responsible for diagnostic acumen in regard to them.

The credit is generally given to Ebner^{3a} for reporting (1904) the first cyst of a semilunar cartilage. Up to about 1930 the lesion was considered as a great rarity and in the minds of many there was confusion as to the differentiation between this lesion and other cystic lesions in this area.

Any tumor in the region of the knee is potentially dangerous and should be removed. It is not always possible to be reasonably confident that a tumor in the region of the lateral or medial meniscus is not a malignant tumor entirely unrelated to the meniscus. Beside the possibility of its being a malignant neoplasm two considerations

make it important to recognize this lesion (1) the fact that the condition practically never corrects itself spontaneously and will continue to disable the patient until it is removed (2) the surgical treatment of a cyst of the meniscus is quite satisfactory, and for this reason it should be undertaken.

INCIDENCE AND LOCATION

A cyst of the meniscus is a rare lesion to encounter. However this remark should be qualified, because a fair percentage of torn external semilunar cartilages which are removed will be found upon close inspection to contain one or more very small cystic lesions which are really cysts of the meniscus. Even so it remains true that the large clinically recognizable lesions are rare. These cysts will be encountered in a large orthopaedic practice from time to time, however if the surgeon keeps on the alert for them.

Cysts of the menisci are most frequently encountered in young men between 18 to 28 years of age. The lesion is found about 10 times more commonly in the lateral meniscus than in the medial. Statistics vary and authors quote a difference of somewhere between 5 to 1 and 10 to 1. The lesion is approximately 10 times more common in men than in women. (These observations support trauma as the responsible etiologic factor; this premise is held by the author.)

In 1928 Bristow^{3a} collected all the cases in the world's literature and found that the total number did not exceed 30 cases. At this time he reported a series of 11 cases

*El Paso, Texas



FIG 1 (Left) A meniscal cyst drawn from an actual specimen showing relation of cystic mass to periphery of the meniscus. (Right) Same specimen sectioned longitudinally showing cystic degeneration in the substance of the meniscus.

Ten years later (1938) Klemberg⁹ reported that only 163 cases had been encountered in the world's literature up to that date. He also stated that few surgeons ever had treated more than one or two cases of this condition. More recently a great many cases have been seen and operated upon. Smillie²⁰ reported a series of 136 personal cases. However some of them were small lesions identified after the removal of the cartilage. Pisanu¹⁷ reported a series of 30 personal cases. McMurray¹³ a series of 28. The author is reporting a series of 18 cases with an interesting clinical curiosity of a photograph of 4 of these under treatment at the same time. A cursory search through the literature at this time by the author reveals that 1,210 cases have been reported, including the 18 cases in his own series. It is possible that some cases in the literature may have been overlooked.

It is of interest to note that Smillie's cases were in workers engaged in heavy industries and that Pisanu's and the author's are in soldiers another occupation requiring vigorous physical activity and frequent exposure to trauma. These series alone strongly support the theory that trauma plays a large part in the production of this lesion.

ETIOLOGY

The etiology is unsettled at the present time. Numerous surgeons and pathologists feel that the lesion is congenital but some competent surgeons feel very strongly that these lesions are traumatic in origin.

Smillie²⁰ a British authority on knee surgery who has reported by far the largest personal series, feels quite strongly that the lesion is traumatic for the following reasons: (1) It is common in young adult males; this is the sociological group which

is exposed most to trauma (2) It is much more common in coal miners than in the average population. (3) It is usually present in the middle third of the cartilage, where most of the trauma to the cartilage occurs.

Zadek and Jaffe²⁴ believe that this lesion is a true cyst and that it is probably congenital in origin. They have written much on the subject supporting this premise.

Ghormley⁶ believes that the lesion is degenerative and not neoplastic. He does feel that it is not a true tumor but represents a degeneration in an area in the semilunar cartilage.

Some workers believe that these lesions are due to multiple minor traumata, superimposed on an inherent tendency in the patient to develop this lesion.

In connection with trauma as an etiologic factor, often it is possible to obtain the history of one relatively severe injury and of repeated minor traumata. In view of this perhaps either type of trauma may be the causative factor in an individual case. The author adheres to this belief, especially since his entire series was found in infantry soldiers engaged in vigorous physical training at a large Army post.

PATHOLOGY

Upon removing the diseased tissue and examining it grossly it is found to be a fibrous mass containing one or more cysts filled with gelatinous material. There may be only one gelatinous cyst; there may be either one large and numerous small ones or many small ones. The gelatinous material which fills the cyst is for all practical purposes the same as that found in a ganglion cyst. It does not have any regular structure. The cyst is attached to the meniscus (Fig. 1).

The pathology of the cyst is somewhat like that of a ganglion. It has been referred to in the past as a ganglion of the outer or inner side of the joint.

These cysts are usually multilocular but occasionally only one specific area is easily seen grossly and the other small areas may

be overlooked. However, sometimes the entire mass consists of numerous small cysts of approximately the same size.

The size of the tumor varies from 1 to 4 cm. in diameter in the cases where it is detectable clinically that is in a typical case of a cyst of the meniscus which is seen as a tumor mass on the outer side of the knee. As stated previously, some of these lesions are identified postoperatively in the meniscus and appear as tiny cystic areas. In the clinically detectable ones they may be pinpoint in size and are barely detectable or they may be the size of a walnut or even larger.

As far as the actual pathogenesis is concerned it is well established that these cysts are degenerative and not neoplastic. There is no endothelial lining present on microscopic examination.

A cyst of the meniscus is located usually in the midportion of the semilunar cartilage and presents clinically anterior to the ligament. Characteristically then, a meniscal cyst will be seen as a tumor mass on the outer side of the knee joint opposite the joint and just in front of the collateral ligament.

There is a considerable difference of opinion as to what percentage of these are associated with tears of the corresponding meniscus. Smillie feels that this lesion is almost always associated with a tear of the cartilage. Part of this difference of opinion is due to the fact that he takes out an external meniscus on the diagnosis of meniscus pathology and with a tear in mind. When he removes the cartilage and finds the tear he then looks carefully for a cyst; often one or more tiny ones are encountered. On the other hand, American authors find that an associated tear of the semilunar cartilage is not a common accompaniment of a meniscal cyst. They operate on a patient with a clinical diagnosis of a cyst of the meniscus and usually find little or no evidence of serious trauma to the meniscus.

There has been much discussion about



FIG. 2. The knee, showing usual site of the tumor mass in relation to the fibular collateral ligament.

the relative importance of differentiating between a parameniscal and a meniscal cyst. Some authors feel quite strongly that there is a difference but Smilie and the author of this paper feel that they are the same and it is simply a matter of location. At operation it is frequently found that a cyst will connect directly with a degenerated area in the meniscus near its periphery and that the cyst may actually originate from this area. Whether the cyst is located in the cartilage proper is closely associated with the periphery or is quite distinct from it and located in a parameniscal area would seem to the author of no great significance since he feels that all of these cysts must have the same origin in

the meniscus itself. It is said that the larger cysts of the menisci are parameniscal and that the smaller ones are usually within the meniscus. This is essentially true, as a large cyst could hardly remain in the meniscus without completely disturbing the joint function. However the author would like to stress again that the differentiation between meniscal and parameniscal cysts is one of differentiating the location and not the etiology and the pathology of the two lesions.

SYMPTOMS

The characteristic symptom of this condition is the complaint of the presence of a mass over the outer or inner side of the knee opposite the joint and at the level of the meniscus (Fig 2). The mass usually presents just in front of the collateral ligament. The presence of a mass is usually the initial chief complaint. The presence of the tumor is of concern to him and brings him to the doctor. As all surgeons know because of the educational program in regard to the early recognition of cancer people are more tumor conscious than formerly and the mere presence of a swelling brings many of them to a doctor's office.

Pain in a varying degree is another symptom present in this condition. Pain varies from a mild discomfort to a relatively severe ache. The discomfort or pain is worse after use. It is also worse on strong flexion or full extension. Characteristically the patient's knee is most comfortable in a position of about 75° of flexion. Occasionally a pinching sensation is noted in the knee in the area where the mass is located.

Sometimes a feeling of giving way of the knee is present but this is not characteristically a symptom of a cyst of the meniscus.

Weakness and easy fatiguing of the knee with this condition sometimes spreading to the entire leg is a complaint usually present. The patient often has trouble in locating the place where the fatigue is worst and simply says that his leg feels weak.

A history of injury is usually given. The

injury may be one definite episode which the patient can recall easily, or a history of repeated minor traumata may be encountered. One or the other or both of these is found in the great majority of cases. The history of one definite injury is present in about one half of the cases of cysts of the menisci.

A characteristic negative consideration in the history is the absence of a story of locking or blocking of the knee. This serves to rule out a bucket handle tear of the meniscus which is of importance in both the diagnosis and the treatment of the lesion.

PHYSICAL EXAMINATION

A characteristic finding in the physical examination is a tumor. It is located exactly opposite the knee joint and at the level of the semilunar cartilage usually in the region of the external semilunar cartilage but on rare occasions in the region of the medial meniscus. The cyst usually presents itself in front of the fibular collateral ligament or the tibial collateral ligament, depending upon whether the cyst is located laterally or medially. The size of the tumor may vary from that of a pea to a hen's egg. The tumor is firm, moderately tense and often has a rubbery feel. There is little difficulty in being reasonably sure that it is a cystic lesion.

One of the most characteristic physical findings in these cases and one which, when present, is considered as pathognomonic by Pisani¹⁵ is the fact that the cyst tends to disappear or decrease greatly in size when the knee is placed in full flexion. He has called this the "disappearing sign."

Tenderness on deep pressure is a characteristic physical finding. There is no redness or heat present, however. Occasionally there is a mild effusion in the knee joint, although this is usually considered to be due to associated pathology. A valuable negative sign is the presence of a normal roentgenogram in these cases.

There is often atrophy of the quadriceps muscle and the calf group; the atrophy is not pronounced in most instances.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of cysts of the menisci is usually easy because of the characteristic location, the "disappearing sign" (the decrease in size of the tumor when the knee is flexed), and because the tumor has the characteristic feel on palpation.

A Baker's cyst must be considered in the differential diagnosis. In rare instances a cyst of the meniscus may project backward toward the popliteal space and in this way pose a difficult differential diagnostic problem. However the characteristic location and feel of a Baker's cyst or bursal cyst usually makes the true diagnosis possible.

Synovial pouches may offer some confusion in the differential diagnosis, but characteristically they are located posteriorly instead of laterally; they extend away from the joint and are not adherent to the semilunar cartilage.

A differential diagnosis of a meniscal cyst from tumors in the region of the knee joint is not always easy. A subcutaneous tumor such as a lipoma or a fibroma, may be puzzling in differentiating it from a meniscal cyst. Its lack of adherence to the cartilage is a most helpful differential point. Ordinarily tumors of the knee joint proper do not extend laterally from the joint and thus in only the rarest instances do they offer a differential diagnostic problem.

TREATMENT AFTER-CARE AND PROGNOSIS

The surgical excision of the cyst, together with the associated meniscus, is the proper treatment in these cases. Formerly it was common practice to excise the cyst only but the prognosis for a satisfactory outcome is not nearly as good as when the entire cartilage is also removed. In the past there has been some difference of opinion as to whether or not it is necessary to excise the entire cartilage or simply the portion from which the cyst originated. It is now well agreed that the entire cartilage should be re-

moved. The exact technic may vary somewhat with the individual operator. It is important to make an incision directly over the cyst for the purpose of freeing it and removing it. The cyst may be removed after identification of all of its parts and its attachments to the underlying cartilage. Then the semilunar cartilage may be removed in the operator's usual way following the removal of the cyst. A somewhat better way to remove both the cyst and the meniscus is by first freeing the cyst by means of an incision made directly over it and then removing the cartilage from before backward through an additional anterior incision. The anterior part of the cartilage is removed back to the region of the cyst, and then the posterior part of the cartilage is removed from the cyst backward the remaining distance. It is often necessary and in fact routinely advised by many authors to make a posterior Henderson incision for removing the back part of the cartilage and making sure that the entire cartilage is excised.

During the removal of the cyst and the cartilage great caution should be exercised not to cut the collateral ligament. If it is cut and not noticed a serious disability results. If it is cut and repaired a longer period of convalescence is required than that usually needed following an uncomplicated arthrotomy and at times a certain degree of permanent disability results.

Recurrence of the tumor is quite rare and the prognosis for a satisfactory result is good.

However the author would like to call attention to the oft repeated statement that the cure is complete and permanent in his opinion that is not quite true. With complete removal of the lateral meniscus a hypertrophic arthritis develops at the outer side of the joint in a fair percentage of cases. Although the degree of hypertrophic arthritis is usually slight and the amount of disability not great it does produce some disability. This disability usually develops late rather than immediately following the opera-

tion and progresses as hypertrophic arthritis develops. These remarks are not to be construed as implying that the meniscus should not be removed, as this is necessary to effect a cure. It is well to caution against telling the patient that the prognosis is extremely good because of the frequent occurrence of hypertrophic arthritis later on.

The after-care of these patients should be the same as that in any other case of removal of a semilunar cartilage from a knee. The after-care varies somewhat with different surgeons although certain things are generally considered desirable. A period of 2 or 3 weeks complete rest is usually advised. Care should be taken to avoid a flexion contracture following the operation. As soon as the immediate postoperative pain has subsided the patient is started on quadriceps strengthening exercises, and in addition to this is given regular physical therapy. Where it is practicable a formal course of rehabilitation is advisable.

As far as the degree of permanent disability is concerned, following the complete removal of the meniscus, the average disability in working men is approximately 10 per cent of the knee joint. The same permanent disability rating will be anticipated in cases following removal of a cyst and its accompanying semilunar cartilage. Occasionally there are cases seen which make a complete recovery and often the disability will be slightly more or slightly less than the 10 per cent figure. Because of this permanent disability the association of trauma with the onset may be of considerable importance in industrial cases. Since trauma is not considered by everyone to be the primary cause of cysts of the meniscus there is room for argument in this regard as to the cause of the cyst in industrial cases. The presence of an associated meniscal tear at operation may settle the issue of the traumatic cause of the patient's condition. Since such tears are not found characteristically the controversial issue may not be settled even after operation.

REPORT OF A SERIES OF 18 CASES OF CYSTS OF THE MENISCI

The author, while in the Army during World War II, was Chief of an Orthopaedic Section at a large regional hospital which was the orthopaedic center for approximately 250 000 troops in training. Most of these were infantry soldiers. Injuries to the knee were relatively common and the author kept a careful count of 400 consecutive arthrotomies of the knee. In this series there were 18 cases of meniscal cyst; all were operated upon, and all obtained a satisfactory result. There was slight disability present at the conclusion of most of these cases. Of the 18 cases 3 had associated meniscal tears. An additional 6 had slight semilunar cartilage damage but not a definite tear.

In the author's 18 cases the majority showed one principal cyst, although in a few multiple cysts were present. An exact tabulation of these was not kept. Most of these cases were apparently parameniscal so far as their exact location was concerned, but in the same cases the cysts appeared to originate from the semilunar cartilages and for

all practical purposes were attached to the meniscus and were an integral part of it in each instance.

The accompanying illustrations (Figs 3 and 4) are of considerable interest, since the photographs show 4 soldiers under treatment at the same time for the same condition. These 4 cases were done within a period of 3 weeks. They represent various periods of convalescence of from 5 to 8 weeks after their operations. This particular group of cases was from a somewhat larger group that occurred in a period of intensive training in an infantry division. All 18 of the cases in the series were engaged in vigorous military training at the time that their cysts developed. In all a history of repeated minor trauma or traumata could be elicited. In most of these one specific injury which was worse than the others could be recalled by the soldier.

Concerning the operation done in these cases, in each instance the entire cartilage was removed as well as the cyst. In the cases with the larger cysts the cyst was removed separately and then the cartilage was removed as a unit usually through 3



FIG 3 Four patients, recently operated upon, within a period of 3 weeks for cysts of the lateral menisci, showing range of flexion. They represent periods of from 5 to 8 weeks after their operations.

incisions. These incisions were located one over the cyst, the second anteriorly in the parapatellar location and the third posteriorly (Henderson incision) for the purpose of getting out the back portion of the cartilage.

As stated above consistently good results were obtained in these cases although slight residual disability was usually encountered. Rehabilitation of these patients was stressed, and the photographs shown were made at a convalescent rehabilitation center. This rehabilitation program speeded up convalescence and reduced the permanent disability.

DISCUSSION

A review of the literature pertaining to the subject of cysts of the menisci together with reflection on the author's series of cases reveals a few points which are worthy of reiteration and comment.

Although this lesion is rare it is of sufficient frequency to require accurate recognition by the clinical orthopaedist. It is essential that an accurate diagnosis be made and adequate treatment be instituted before these

lesions can be readily eradicated and the patients relieved of their symptoms.

Although pathologically it may not be possible to establish the causal relation between trauma and the lesion, nevertheless it is clinically apparent that such a relationship exists. The authors who have reported the largest series of cases have found their patients to be in groups of individuals whose knees are subjected to trauma.

CHART I. STATISTICAL ANALYSIS OF A SERIES OF 18 MENISCAL CYSTS ENCOUNTERED IN 400 CONSECUTIVE KNEE ARTHROTOMIES

Number of cases	18
Number of cases operated upon	18
Good result	18
Poor result	0
Average residual disability	10%
Average time before return to work	10 weeks
Number of cases with associated meniscal tears	3
Number of cases with history of preceding trauma	18
Number of cases in which the meniscus was removed as well as the cyst	18



FIG. 4 The same four patients as in Figure 3 but here showing the range of extension, which is complete. Strong quadriceps power is present.

The characteristic thing about cysts of the menisci is that they usually occur in young vigorous males between the ages of 18 and 28 and they do not occur with any degree of frequency in other classes of people. This will further substantiate the theory that trauma is causative.

The author's series of cases is typical in most respects and shows that a group of healthy young adults who are engaged in vigorous physical training will show a relatively high incidence of cysts of the menisci.

SUMMARY

1 Cysts of the menisci, although rare occur with sufficient frequency to warrant serious consideration by the orthopaedic surgeon.

2 All phases of the object are reviewed and discussed, including etiology, pathology, diagnosis and treatment.

3 The typical patient with a cyst of a meniscus is a young adult male with an occupation involving considerable physical activity who subjects his knee to repeated minor traumata.

4 Cysts of the menisci arise much more commonly from the external meniscus.

5 These cysts occur characteristically in the central portion of the lateral meniscus and present themselves clinically in front of the fibular collateral ligament.

6 Treatment should be by excision of the cyst and the entire semilunar cartilage associated with it.

7 This treatment gives consistently good results although slight disability may be anticipated. Recurrences are practically unknown.

8 Orthopaedic surgeons in general consider these cysts to be of traumatic origin. Some pathologists think that they are of congenital origin. They are generally considered to be degenerative and not neoplastic.

9 The author has presented a series of 18 cases of cysts of the menisci occurring in 400 consecutive arthrotomies of the knee, all of which were operated upon and all of

which had a satisfactory end result. These cases occurred in troops in training in World War II.

REFERENCES

1. Bennett G. E. Cysts of the semilunar cartilage. *Am J Surg.* 32:512, 1939.
2. Brav E. A. Cysts of the semilunar cartilages of the knee joint. *Mil Surgeon* 105:278, 1949.
3. Casacci A. Bilateral and symmetric cysts of external meniscus of knee. *Ateneo par mense* 19:94, 1948.
- 3a. Bristow W. Rowley. Cysts of the semilunar cartilages of the knee. *Robert Jones Birthday Volume* p. 269. London: Oxford, 1928.
4. Dittich R. J. Concealed cysts of the lateral meniscus of the knee. *J Bone & Joint Surg.* 28:646, 1946.
5. Elskind, L. Semilunar cartilage cysts. *Acta. orthop. scandinav.* 8-9:317, 1938.
- 5a. Ebner A. Ein Fall von Ganglion am Kniegelenksmeniskus. *München. med. Wchnschr.* 51:1737, 1904.
6. Ghormley R. H. and Dockerty M. B. Cystic myxomatous tumors about the knee: their relation to cysts of the menisci. *J Bone & Joint Surg.* 25:306, 1943.
7. Grosic, F. Popliteus muscle and cysts of external meniscus of knee: syndrome of popliteus. *Rev. orthop.* 36:543, 1950.
8. King, E. S. J. Formation of ganglia and cysts of the menisci of the knee. *Surg., Gynec. & Obst.* 70:150, 1940.
9. Kleinberg, S. Cysts of external semilunar cartilage. *Arch. Surg.* 37:827, 1938.
10. Kulowski, J. Meniscus cyst. *J. Missouri M. A.* 37:503, 1940.
11. Kulowski, J., and Rickett, H. W. The relation of discoid meniscus to cyst formation and joint mechanics. *J Bone & Joint Surg.* 29:990, 1947.
12. Leemans. Relation between cysts of meniscus and cystic formation of conjunctival origin. *J. chir. et. Ann. Soc. belge de Chir.* 36-38:290, 1939.
13. Lewin, P. Cysts of the menisci, from *The Knee and Related Structures*, p. 235. Philadelphia, Lea & Febiger, 1952.
14. McDermott, L. J. Cysts and degenerative changes in the menisci of the knee joint. *J. Maine M. A.* 43:116, 1952.
15. McReynolds, I. S. Cysts of semilunar cartilages. *South. M. J.* 32:571, 1939.

- 16 Norrander E. Semilunar cartilages, cysts and ganglion *Acta orthop scandinav* 7 362, 1936
- 17 Nunziata, A. Semilunar cartilages-cysts, *Prensa méd. argent.* 31 1205 1944
- 18 Pisani A. J. Pathognomonic sign for cyst of the knee cartilage *Arch. Surg.* 54 188 1947
- 19 Ransohoff N W A cyst of the lateral meniscus causing peroneal nerve palsy *Bull. Hosp Joint Dis* 2.69 1941
- 20 Smillie, I S Injuries of the Knee Joint, pp 48-53 and 89-93 Baltimore, Williams & Wilkins, 1951
- 21 Vidal Naquet, G Cysts of external meniscus-surgical therapy of case *Bull. et mém. Soc. chir Paris* 51 137 1939
- 22 Willard DeF P and Nicholson J T Cyst of the semilunar cartilage, *Ann. Surg* 112.305 1940
- 23 Wolff H. Semilunar cartilages-cysts clinical study *Chir org movimento* 23 18 1937
- 24 Zadek, I and Jaffe H L. Cysts of semilunar cartilages of the knee *Arch. Surg.* 15 677 1927

Traumatic Lesions of Menisci

R A MURRAY, M D *

Traumatic lesions of menisci have been recognized and treated under the general term "internal derangement of the knee" since the eighteenth century when William Hay⁷ described this condition. Although there has been much improvement in methods of diagnosis and surgical technique differences of opinion still exist regarding the proper care of the patient with a mechanically imperfect knee^{6,12}. There is general uniformity of opinion regarding the desirability of removing a chronically defective meniscus; however many conditions can mimic the "torn cartilage" and a careful investigation is necessary to make a definite clinical diagnosis. A high percentage of exploratory operations with a surgically lacerated meniscus as the only pathologic specimen may be compared with the spine operation with removal of a nonpathologic ligament or cartilage. Both are frequently rewarded by the persistence of complaints.

ANATOMY AND PATHOLOGY

Menisci are functioning parts of a mechanical, movable joint and naturally are subjected to stress and strain that may tear, dislocate or relax any portion of them. Situated at the margin of the joint, they fill an otherwise dead space between the periphery of the condyles of the tibia and the femur keeping the capsule and the synovia from

becoming pinched between the joint surfaces. Functionally, they cushion hyperflexion and hyperextension of the knee, compensate for inequalities of the joint surfaces, diminish friction and participate in all knee motions⁴ (Figs 1 and 2).

It has been estimated that traumatic lesions of the medial meniscus occur with 8 to 10 times the frequency of lateral meniscus injuries. Primarily this is attributed to the more frequent use of the medial pivot mechanism that produces the medial meniscus injury and to its more firm attachment to the capsule. Bucket handle tears are the most frequent injuries giving the classic symptoms and findings; however anterior or posterior horn lacerations or any combination of tears may be found (Figs 3 and 4).

Meniscus injuries are often associated with other traumatic lesions of the knee. Cruciate or collateral ligament strains or ruptures are frequently accompanied by meniscus tears. Fractures of the articulating ends of the femur and the tibia may also traumatize the menisci. The tibial plateau fracture is associated most commonly with meniscus ruptures and when open reduction of this fracture is undertaken, the menisci, if disorganized, should be excised. We believe however that, if normal it is important not to lacerate the meniscus surgically so that removal is necessary but to attempt to preserve the structure for its cushioning effect in partially leveling a potentially irregular joint surface.

*Department of Orthopedic Surgery of the Scott and White Clinic, Temple, Texas.

Jack⁸ has called attention to a lesion of the attachment of the lateral meniscus. The peripheral attachment of the lateral meniscus differs from the medial in that it is interrupted by the passage of the femoral tendon of the popliteus muscle. The lesion is a tear of the synovial reflection and the coronary ligaments backward from this tunnel so that the posterior half of the cartilage loses its attachment peripherally and slips forward into the joint. The onset usually follows a period of relaxation with the knees completely flexed, as after squatting on the heels. The symptoms are locking and postero-lateral knee pains with the lock occurring in more than normal flexion often 90° and can be released easily by straight traction. Removal of these menisci is simplified because of the posterior detachment.

Kings^{9,10} experimental work on dogs and reported cases on humans¹¹ demonstrate that the excised meniscus is replaced by a new functioning meniscuslike fibrous structure that may be injured again. Articular hyaline cartilage degeneration occurs over the joint surfaces proportionate to the excised segment after meniscectomy. These degenerative changes are minimal, however

when compared with the traumatic arthritis that develops if a locked or torn cartilage is allowed to remain in a functioning joint.

SYMPTOMATOLOGY AND PHYSICAL FINDINGS

The classical signs and symptoms of an injured meniscus are not pathognomonic, as they may be reproduced by a host of other lesions of the knee. Adequate history, physical findings and routine anteroposterior, lateral and intercondylar notch roentgenograms must all be evaluated to differentiate these lesions. Typically a history is given of an injury sustained to the flexed, weight bearing knee when it is twisted as by a pivot (internal rotation of the femur on the tibia). The meniscus, which is fixed between the tibial and the femoral condyles, tears as excessive rotation occurs. Acute pain results, followed by swelling secondary to hemarthrosis or synovial fluid effusion. The knee may lock immediately so that complete extension or flexion is impossible or activity may be continued without marked discomfort until a period of rest. Limited knee motion may occur secondary to interposition of the torn cartilage or joint distention,

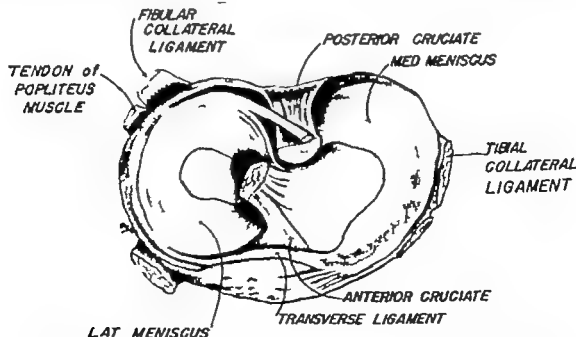


FIG. 1 Articulating tibial surface with menisci and ligaments (Redrawn from Grant's *Atlas of Anatomy*)

the joint capsule is the most relaxed at to 30° of flexion. Extension is limited by a tear of the anterior horn or bucket-handle tear while a posterior horn tear usually results in limitation of flexion. Pain and tenderness are present over the cartilage and may be so acute that adequate motion is impossible. Occasionally, the first symptoms of damage are less dramatic and it is usual for the "repeaters" to have considerably less acute findings. True locking is considered pathognomonic if no body or other roentgenographic abnormality is visible. It is of the utmost importance to establish the mechanism of injury—what the patient means by locking, the thrusts, instability getting out of joint, etc.

Careful examination of the knee should include palpation of the entire joint area, including the popliteal surface, the suprapatellar pouch, the joint line, the patella and the patellar tendon insertion. Stability of the cruciate and the collateral ligaments should be tested. Tenderness over the cartilage is an important sign when correlated with history and other findings.

Adequate examination is especially important when discomfort is acute following a fresh injury for collateral and cruciate ligament damage must be considered. Aspiration and procaine injection will usually allow a relatively painless examination to determine if a ligament has been torn. If there is still question, we think that it is best to anesthetize the patient and be prepared to repair the injured ligament surgically if instability is found. Although we may procrastinate and treat an injured meniscus conservatively, a severely torn ligament deserves an early adequate surgical repair.¹⁰ Late repairs of damaged ligaments are not usually satisfactory while meniscectomy may be delayed, if there is a question as to the diagnosis, without increasing the difficulty or jeopardizing the success of the surgical procedure.

Manipulation of the knee to reproduce the pain and, if possible, a click or palpable

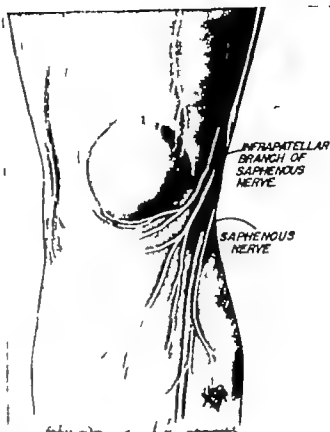


Fig. 2 Infrapatellar branch of saphenous nerve



Fig. 3 Medial meniscectomy surgical specimen—old transverse and marginal tear

sensation over the cartilage is of help. There are several tests all using the principle of reproduction of the mechanism of injury (external or internal rotation of the tibia on the femur) which vary in whether the extremity is weight bearing or is being carried through a range of motion. The physician should become familiar with these and use all of them or his favorite of those described by Apley,³ McMurray,¹¹ Presbyte,¹² or the Hospital group,³ etc. They all depend

Jack⁸ has called attention to a lesion of the attachment of the lateral meniscus. The peripheral attachment of the lateral meniscus differs from the medial in that it is interrupted by the passage of the femoral tendon of the popliteus muscle. The lesion is a tear of the synovial reflection and the coronary ligaments backward from this tunnel so that the posterior half of the cartilage loses its attachment peripherally and slips forward into the joint. The onset usually follows a period of relaxation with the knees completely flexed, as after squatting on the heels. The symptoms are locking and postero-lateral knee pains with the lock occurring in more than normal flexion often 90° and can be released easily by straight traction. Removal of these menisci is simplified because of the posterior detachment.

King's^{9,10} experimental work on dogs and reported cases on humans¹¹ demonstrate that the excised meniscus is replaced by a new functioning meniscuslike fibrous structure that may be injured again. Articular hyaline cartilage degeneration occurs over the joint surfaces proportionate to the excised segment after meniscectomy. These degenerative changes are minimal however

when compared with the traumatic arthritis that develops if a locked or torn cartilage is allowed to remain in a functioning joint.

SYMPTOMATOLOGY AND PHYSICAL FINDINGS

The classical signs and symptoms of an injured meniscus are not pathognomonic, as they may be reproduced by a host of other lesions of the knee. Adequate history, physical findings and routine anteroposterior, lateral and intercondylar notch roentgenograms must all be evaluated to differentiate these lesions. Typically a history is given of an injury sustained to the flexed, weight bearing knee when it is twisted as by a pivot (internal rotation of the femur on the tibia). The meniscus, which is fixed between the tibial and the femoral condyles, tears as excessive rotation occurs. Acute pain results followed by swelling secondary to hemarthrosis or synovial fluid effusion. The knee may lock immediately so that complete extension or flexion is impossible or activity may be continued without marked discomfort until a period of rest. Limited knee motion may occur secondary to interposition of the torn cartilage or joint distention,

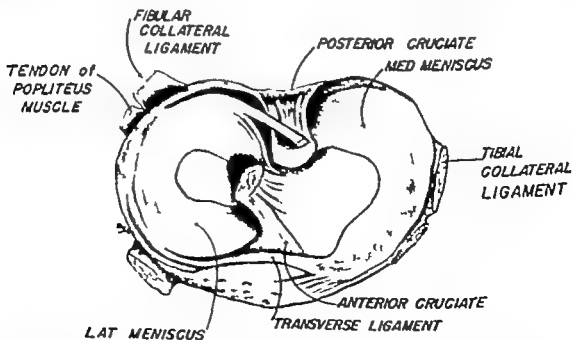


FIG. 1 Articulating tibial surface with menisci and ligaments (Redrawn from Grant's *Atlas of Anatomy*)

for the joint capsule is the most relaxed at 20 to 30° of flexion. Extension is limited by a tear of the anterior horn or bucket-handle tear, while a posterior horn tear usually results in limitation of flexion. Pain and tenderness are present over the cartilage area and may be so acute that adequate examination is impossible. Occasionally the first symptoms of damage are less dramatic and it is usual for the "repeaters" to have considerably less acute findings. True locking is considered pathognomonic if no loose body or other roentgenographic abnormality is visible. It is of the utmost importance to establish the mechanism of injury—what the patient means by locking, clicking, thuds, instability getting out of joint, etc.

Careful examination of the knee should include palpation of the entire joint area, including the popliteal surface, the suprapatellar pouch, the joint line, the patella and the patellar tendon insertion. Stability of the cruciate and the collateral ligaments should be tested. Tenderness over the cartilage is an important sign when correlated with history and other findings.

Adequate examination is especially important when discomfort is acute following a fresh injury for collateral and cruciate ligament damage must be considered. Aspiration and procaine injection will usually allow a relatively painless examination to determine if a ligament has been torn. If there is still question, we think that it is best to anesthetize the patient and be prepared to repair the injured ligament surgically if instability is found. Although we may procrastinate and treat an injured meniscus conservatively, a severely torn ligament deserves an early adequate surgical repair.¹² Late repairs of damaged ligaments are not usually satisfactory while meniscectomy may be delayed, if there is a question as to the diagnosis, without increasing the difficulty or jeopardizing the success of the surgical procedure.

Manipulation of the knee to reproduce the pain and, if possible, a click or palpable

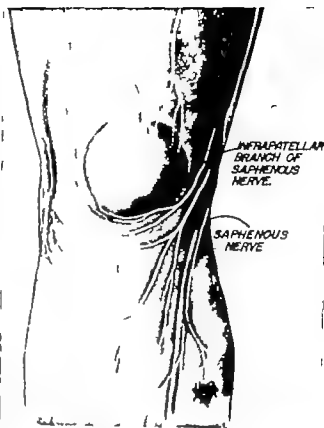


FIG 2 Infrapatellar branch of saphenous nerve.



FIG 3 Medial meniscectomy surgical specimen—old transverse and marginal tear

sensation over the cartilage is of help. There are several tests all using the principle of reproduction of the mechanism of injury (external or internal rotation of the tibia on the femur) which vary in whether the extremity is weight bearing or is being carried through a range of motion. The physician should become familiar with these and use all of them, or his favorite of those described by Apley,² McMurray,¹¹ Presbysman Hospital group³ etc. They all depend

Jack⁵ has called attention to a lesion of the attachment of the lateral meniscus. The peripheral attachment of the lateral meniscus differs from the medial in that it is interrupted by the passage of the femoral tendon of the popliteus muscle. The lesion is a tear of the synovial reflection and the coronary ligaments backward from this tunnel so that the posterior half of the cartilage loses its attachment peripherally and slips forward into the joint. The onset usually follows a period of relaxation with the knees completely flexed after squatting on the heels. The lesion is locking and posterolateral in position with the lock occurring in maximal medial flexion, often 90° and can be released easily by straight traction. Removal of these menisci is simplified because of the posterior detachment.

Kings⁶ experimental work on dogs and reported cases on humans^{7,8} demonstrate that the excised meniscus is replaced by a new functioning meniscuslike fibrous structure that may be injured again. Articular hyaline cartilage degeneration occurs over the joint surfaces proportionate to the excised segment after meniscectomy. These degenerative changes are minimal however

when compared with the traumatic arthritis that develops if a locked or torn cartilage is allowed to remain in a functioning joint.

SYMPTOMATOLOGY AND PHYSICAL FINDINGS

The classical signs and symptoms of an injured meniscus are not pathognomonic, as they may be reproduced by a host of other lesions of the knee. Adequate history physical findings and routine anteroposterior lateral and intercondylar notch roentgenograms must all be evaluated to differentiate these lesions. Typically a history is given of an injury sustained to the flexed, weight-bearing knee when it is twisted as by a pivot (internal rotation of the femur on the tibia). The meniscus which is fixed between the tibial and the femoral condyles, tears as excessive rotation occurs. Acute pain results, followed by swelling secondary to hemarthrosis or synovial fluid effusion. The knee may lock immediately so that complete extension or flexion is impossible or activity may be continued without marked discomfort until a period of rest. Limited knee motion may occur secondary to interposition of the torn cartilage or joint distention.

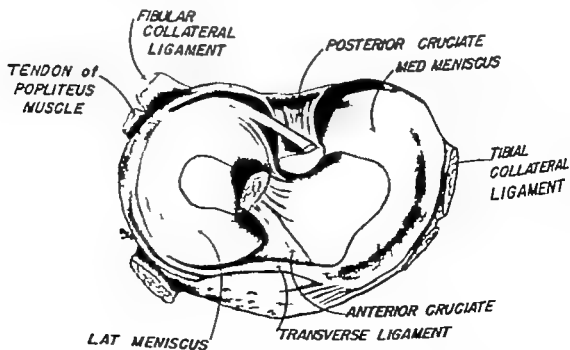


FIG. 1 Articulating tibial surface with menisci and ligaments (Redrawn from Grant⁹)

for the joint capsule is the most relaxed at 20 to 30° of flexion. Extension is limited by a tear of the anterior horn or bucket handle tear while a posterior horn tear usually results in limitation of flexion. Pain and tenderness are present over the cartilage area and may be so acute that adequate examination is impossible. Occasionally, the first symptoms of damage are less dramatic and it is usual for the "repeaters" to have considerably less acute findings. True locking is considered pathognomonic if no loose body or other roentgenographic abnormality is visible. It is of the utmost importance to establish the mechanism of injury—what the patient means by locking, clicking, thuds, instability getting out of joint, etc.

Careful examination of the knee should include palpation of the entire joint area, including the popliteal surface, the suprapatellar pouch, the joint line, the patella and the patellar tendon insertion. Stability of the cruciate and the collateral ligaments should be tested. Tenderness over the cartilage is an important sign when correlated with history and other findings.

Adequate examination is especially important when discomfort is acute following a fresh injury for collateral and cruciate ligament damage must be considered. Aspiration and procaine injection will usually allow a relatively painless examination to determine if a ligament has been torn. If there is still question, we think that it is best to anesthetize the patient and be prepared to repair the injured ligament surgically if instability is found. Although we may procrastinate and treat an injured meniscus conservatively, a severely torn ligament deserves an early adequate surgical repair.¹² Late repairs of damaged ligaments are not usually satisfactory while meniscectomy may be delayed if there is a question as to the diagnosis without increasing the difficulty or jeopardizing the success of the surgical procedure.

Manipulation of the knee to reproduce the pain and, if possible, a click or palpable

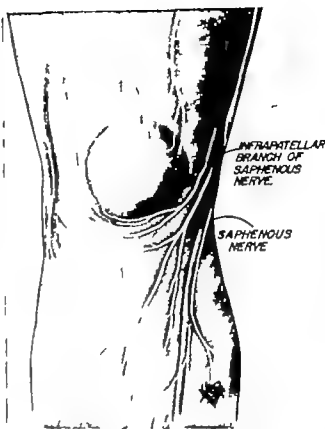


FIG 2. Infrapatellar branch of saphenous nerve.



FIG 3. Medial meniscectomy surgical specimen—old transverse and marginal tear.

sensation over the cartilage is of help. There are several tests all using the principle of reproduction of the mechanism of injury (external or internal rotation of the tibia on the femur) which vary in whether the extremity is weight bearing or is being carried through a range of motion. The physician should become familiar with these and use all of them or his favorite of those described by Apley,³ McMurray,¹¹ Presbyte,¹⁰ Hospital group,³ etc. They all depend

upon the production of a click, a palpable thud, or pain over the menisci as a positive test. Apley² recommends examination with the patient lying prone. The foot is grasped and rotated externally while the knee is flexed completely. The foot is then rotated internally and the knee is extended noting the range of motion and the arc of painful rotation. Next, the examiner's knee is placed on the patient's thigh to fix the upper leg to the table while distraction by a pull on the foot and external rotation of the knee are carried out to demonstrate rotation strain. Lastly the surgeon leans on the foot for compression of the knee while it is rotated. Apley states that if this part of the maneuver is painful it indicates a positive grinding test and meniscus damage.

The late findings are recurrent locking, clicking, or a thud felt over the meniscus area on knee motion, pain and local tenderness recurrent or persistent effusion, a feeling of insecurity, quadriceps atrophy and limitation of extension of a few degrees. At times very few of these findings will be present. If roentgenograms are negative and discomfort is localized to the meniscus area, the knee must be explored because it is the cause of persistent disability.

Recently Smith and Blair¹⁸ have called attention to those patients who have the symptoms and the findings of a medial liga-

ment strain. Pain and tenderness are present on weight bearing, especially with the knee flexed and with sudden external rotation of the tibia on the femur. Trauma, locking, or swelling may not be associated with the symptoms. Conservative treatment, without relief, should certainly precede any surgical exploration and removal of the meniscus. However in this type of patient, persistent disability in spite of adequate treatment, justifies operative exploration. If caution and judgment are exercised, excellent results can be anticipated.

We do not believe that pneumoroentgenograms are of sufficient diagnostic help to justify their routine use; however, roentgenograms including anteroposterior, lateral and intercondylar notch views, should be used routinely. Occasionally there will be slight joint narrowing on the effected side. The diagnosis of a torn meniscus partially depends on normal roentgenographic findings; however it is possible to have a lesion visible in the roentgenogram associated with an abnormal meniscus. Symptoms localized to this area should not be ignored in the treatment of the roentgenographically visible lesion. At times, both menisci will be torn and should be removed. Occasionally it may be difficult to determine preoperatively which cartilage is producing symptoms.

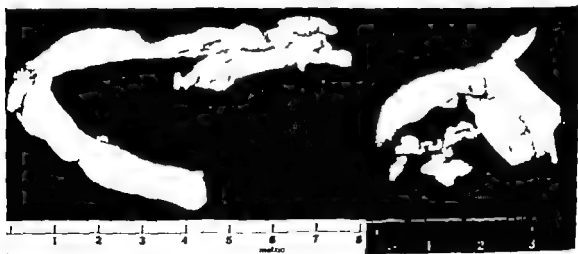


FIG. 4 (Left) Medial meniscectomy surgical specimen—old bucket handle tear
FIG. 5 (Right) Surgical specimen of posterior portion of medial meniscus, giving symptoms following previous incomplete removal

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes most lesions of the knee

A. Nontraumatic Lesions of Menisci

- 1 *Discoid cartilage*—usual lateral more frequent in females, symptoms of snapping or popping on extension may cause pain, lock etc
- 2 *Cysts*—usual lateral cartilage more frequent in females symptoms as discoid, but mass may be felt originating from the meniscus
- 3 *Calcified cartilage*—roentgenographic diagnosis only, may produce no symptoms unless injured
- 4 *Hypertrophied or mobile*—same symptoms and findings as a torn meniscus

B. Common Lesions with Similar Symptoms

- 1 *Rotation strain*—Apley's test, absence of locking clicking or thud
- 2 *Osteochondritis dissecans*—visible roentgenographically
- 3 *Patellar chondromalacia*—patella femoral pressure causing grating and discomfort
- 4 *Osteochondromatosis*—visible roentgenographically and often palpable
- 5 *Collateral ligament bursitis*—tenderness over bursa area, lack of lock or click
- 6 *Collateral or cruciate ligament strain rupture or calcification*—instability on examination, visible roentgenographically on strain
- 7 *Hypertrophied or pedunculated fat pads*—indistinguishable
- 8 *Synovitis*—history and findings
 - a. Traumatic
 - b. Inflammatory
- 9 *Hypertrophic or traumatic arthritis with spurs from the articular surfaces of the tibia or the femur*—visible roentgenographically

- 10 *Loose bodies from patella synovia or articular surfaces of femur or tibia*—palpable or visible roentgenographically
- 11 *Cruciate ligament injuries with stumps projecting into joint*—examination
- 12 *Foreign bodies*—visible roentgenographically, plus history
- 13 *Snapping knee*—no disability or positive physical findings

C. Unusual Lesions Producing Knee Symptoms That Rarely Should Be Confused

- 1 *Xanthomas hemangiomas synoviomias or other tumors of the synovium*
- 2 *Tumors of the distal femur or proximal tibia*

D. Lesions About the Knee That Should Not Be Confused

- 1 *Baker's cyst and other bursa involvements about the knee*
- 2 *Osgood-Schlatter disease*
- 3 *Arthritides such as gout low-grade rheumatoid arthritis etc*
- 4 *Tubercular or gonorrheal arthritis*
- 5 *Charcot knee*
- 6 *Intermittent hydrops*
- 7 *Dislocation of the patella*—acute or chronic

CONSERVATIVE TREATMENT

Conservative treatment of meniscus injuries should be considered only for the acute first episode or when the diagnosis is questionable. Tears through the menisci proper cannot heal due to avascularity while those at the periphery where connective tissue and blood supply are present, heal by fibrous rather than cartilaginous union.⁹ When the diagnosis is definite with typical findings, immediate removal of the torn menisci, even with the first injury has much in its favor. Continued use of a locked knee produces severe cartilaginous joint surface change that can lead only to an irreparable, traumatic arthritis.

The first step in the conservative treatment of the acute episode is aspiration of the joint, for hemarthrosis or effusion lead to chronic synovitis quadriceps atrophy and joint relaxation. Routine surgical skin preparation is warranted to prevent infection. Local procaine anesthesia will allow the painless insertion of a large bore needle either medial or lateral to the mid-point of the patella with the point directed under the superior patella. Pressure on the supra-patellar pouch and the popliteal space allows more nearly complete removal of the fluid. If there is no question of infection the instillation of hydrocortisone 1 cc prevents further effusion or synovial reaction and gives more nearly complete relief of discomfort. We have experienced no complications locally or systemically with its use in this condition and believe that, clinically it has been of great help.

If the knee is locked and aspiration has not restored free motion an attempt may be made to reduce the displaced meniscus by manipulation. While in the supine position the patient's hip and knee are flexed. The lower leg is abducted on the femur and suddenly extended while being held internally rotated. This should be attempted with the extremity relaxed but, if unsuccessful, the patient may help while sitting erect, by actively kicking during the maneuver. If reduction does not occur Buck's traction may be attempted. Reduction under anesthesia has been recommended however if an anesthetic is to be given, it would seem to be more reasonable to remove the meniscus at that time.

Following aspiration and reduction, a well-padded pressure dressing and ice bags for 24 hours, are followed by the application of an ankle-to-groin plaster cast. To prevent the cast from riding on the ankle adhesive tape straps the length of the leg and turned back and incorporated in the cast, suspend the cast from the entire leg.

Quadriceps exercises are a most important factor in rehabilitation of any symptomatic knee and for best results they should be begun prior to cast application. It should

be stressed that exercising the quadriceps is the only way the patient can actively help in his own care. This constant exercising preserves the muscle strength and size. Frequently it may be extremely difficult for the patient to learn to contract the muscle. Setting of the quadriceps of both extremities, electrical stimulation and patience should be employed, if necessary until active contraction is possible. The patient is advised to contract the muscle at least 5 to 10 minutes out of each hour while awake. The plaster cast is worn for 3 to 6 weeks during which time the patient is ambulatory. Following its removal, quadriceps resistant exercises are stressed. If symptoms recur the surgical removal of the menisci is advised.

SURGICAL TREATMENT

Removal of a meniscus may be accomplished in any number of ways. There is such variation in the different techniques that have been recommended that all they have in common is the removal of some meniscus tissue through an incision. The usual 48 hour orthopedic preparation may be used. We prefer a 10-minute soap or hexachlorophene soap-and water scrub followed by ether alcohol, ether and antiseptic immediately before operation. We never have had a postoperative infection in a knee. We prefer a pneumatic tourniquet to secure a bloodless field but have no argument with those not favoring its use. Our anesthetic choice is thiopental sodium and nitrous oxide or spinal anesthesia and our position of choice is with the knee bent and draped so the sterile foot rests on the operator's lap (the operator in sitting position). Abbott's¹ description of surgical approaches to the knee should be consulted by those desiring choice of incisions and exposures. The short median or lateral prepatellar incision has the advantage that it may be extended easily for adequate exposure if an unexpected lesion is encountered. With any exposure care should be exercised to avoid injury to the infrapatellar branch of the saphenous nerve or excessive trauma to the synovium (Fig. 2). Following incision of

the synovium the meniscus is inspected to verify the diagnosis (a posterior tear will not be visible). A routine method of examination of the joint should be employed to avoid overlooking other lesions (C. R. Murray¹² has stated that in his experience 70 per cent of knees opened for torn menisci had associated pathology). This routine should include palpation or direct vision of the posterior patella, the suprapatellar pouch, the fat pad, the cruciate ligaments, the joint surface and if possible, a limited view of the other meniscus. If the meniscus appears to be normal, or if other lesions are found, the incision should be lengthened to allow more adequate exposure and treatment. Severance of the ligamentum mucosum and removal of a portion of the fat pad allow more adequate examination. If symptoms and findings indicate that the meniscus is torn and if no other lesion is found, the meniscus should be removed for the lesion probably will be in the invisible posterior horn. To remove the meniscus the anterior horn is severed from the anterior transverse ligament and grasped with Kocher's forceps. A knife cartilage scissors or special meniscus knife (Smillie¹³ Lowe Breck, etc.) then may be used to dissect the meniscus from the coronary ligament. Occasionally the cartilage may be pulled through the intercondylar notch and the posterior attachment severed to allow complete removal through the one anterior incision. It may be impossible to remove the entire meniscus in this manner without excessive trauma to the joint surfaces. In our experience it is important to remove the entire meniscus and, therefore a second posterolateral or posteromedial incision is used to allow complete removal with direct vision (Fig. 5). The collateral ligaments must be protected throughout the procedure. We do not believe that excising only the free margin of a bucket handle tear is sufficient, because the remaining portion may contain hidden tears and may be more unstable than the fibrous tissue that will regenerate and replace it. Following cartilage removal, the tourniquet may be deflated to allow com-

plete hemostasis. We usually tie all visible bleeders during the procedure, then close the wound in layers, apply a voluminous pressure dressing from toes to groin and a posterior plaster splint before tourniquet removal. After closure of the synovium hydrocortisone 1 cc. is instilled into the knee. We believe that this one point in technic has done more to accomplish a smooth postoperative course than any other that we employ. There is less postoperative discomfort, and effusion sufficient to justify aspiration is infrequent. Apparently due to lack of pain, quadriceps strength is regained more quickly. We have used this drug postoperatively on two patients who previously had torn menisci removed from the opposite knee and these patients inquired why the second procedure was so much less painful than the first. We have not used hyaluronidase in this manner as hydrocortisone has been satisfactory. Postoperatively penicillin is given parenterally but not into the knee at the time of surgery.

Postoperative treatment varies with the patient's course of recovery. It is important to start quadriceps exercises as soon as possible following surgery. The leg is elevated for 24 hours, and then the patient is allowed to position the extremity as desired. The pressure dressing is removed on the third day and if quadriceps muscle power is returning, crutch walking is allowed when the patient desires. It is recommended that crutches not be discarded until quadriceps muscle strength is approaching normal. If postoperative effusion does develop after the third day aspiration is performed with injection of hydrocortisone into the knee. This rarely need be repeated. The most important part of the postoperative routine is the rebuilding of the quadriceps muscle. Resistive exercises must be practiced until atrophy and weakness have disappeared.

PROGNOSIS AND COMPLICATIONS

All surgical procedures are liable to a percentage of unsatisfactory results. This may be due to improper selection of patients or inadequate surgical judgment and technic.

It is our obligation to keep this percentage at a minimum. When treating a benign condition, such as a torn meniscus we should give a patient a reasonably accurate prognosis and make every attempt to accomplish a result better than that anticipated. Although we may dispel the patient's fear of a stiff knee following surgery other factors must be recognized. Those conditions militating against an excellent result (although at times still not contraindicating the procedure) are

A. Late Removal of a Torn Meniscus After Joint Changes Have Already Occurred

B. Associated Pathology That Cannot Be Completely Remedied

- 1 Torn or relaxed cruciate or collateral ligaments
- 2 Associated chondromalacia
- 3 Traumatic or degenerative arthritis and chronic synovitis
- 4 Inadequate personality — compensation
- 5 A patient over 40 years of age
- 6 Flexion contracture of the knee joint

C. Complications of Imperfect Surgery or Uncontrolled Factors

- 1 Rough surgery with injury to joint surfaces ligaments or soft tissue
- 2 Severance of infrapatellar nerve
- 3 Postoperative aneurysm of the inferior lateral geniculate artery³
- 4 Popliteal artery injury¹⁴
- 5 Infection
- 6 Incomplete removal — usually the posterior horn
- 7 Tourniquet paralysis — nonpneumatic tourniquet over areas of nerve vulnerability

D. Inadequate Quadriceps Exercises and Follow Up With Resulting Flexion Contracture and Quadriceps Atrophy

E. Diagnostic Error

F. Postoperative Phlebitis

SUMMARY

Traumatic lesions of menisci are frequently the cause of knee joint disability. Differential diagnosis includes most pathologic lesions of the knee. Conservative treatment is indicated only with the first symptomatic episode or when diagnosis is questionable. Surgical removal of the entire cartilage is indicated with recurrence of symptoms. Quadriceps exercises are of great importance as with all other knee joint pathology, in conservative and operative treatment of meniscus injuries. Injection of hydrocortisone is the only major improvement in recent years for conservative or surgical treatment of meniscus lesions.

REFERENCES

- 1 Abbott, L. C. and Carpenter W. F. Surgical approaches to the knee joint, *J Bone & Joint Surg* 27:277 1945
2. Apley, A. G. The diagnosis of meniscus injuries *J Bone & Joint Surg* 29:78 1947
- 3 Bancroft, F. W., and Marble, H. C. Surgical Treatment—Motor Skeletal System, ed. 2, pt. 2, p 1095 Philadelphia, Lippincott, 1951
- 4 Brantigan, O. C. and Voshell, A. F. The mechanics of the ligaments and menisci of the knee joint, *J Bone & Joint Surg.* 23:44 1941
- 5 Fairbank, T. J., and Jamieson E. S. A complication of lateral meniscectomy *J Bone & Joint Surg* 33-B:567 1951
- 6 Fisher, A. G. Timbrell Internal Derangements of the Knee-Joint, ed. 2 London, Lewis, 1933
- 7 Hay William cited by Fisher⁶
- 8 Jack, E. A. Posterior peripheral detachment of lateral cartilage *J Bone & Joint Surg.* 35-B:396, 1953
- 9 King, Don. The healing of semilunar cartilages, *J Bone & Joint Surg.* 18:333 1936
- 10 ——— The function of semilunar cartilages, *J Bone & Joint Surg* 18:1069 1936.
- 11 McMurray, T. P. The semilunar cartilages, *Brit. J Surg.* 29:407 1942.
12. Murray C. R. Complicating factors in the treatment of injuries to the menisci of the knee joint, *Am. J Surg.* 55:262 1942.
- 13 O'Donoghue, D. H. Surgical treatment of

- fresh injuries to the major ligaments of the knee J Bone & Joint Surg. 32 A 721 1950.
- 14 Ross, W T Injury to the popliteal artery during meniscectomy J Bone & Joint Surg. 33-B 571 1951
- 15 Smillie I S Injuries of the knee joint, Baltimore Williams & Wilkins, 1946
- 16 Smith F B and Blair H C Tibial collateral ligament strain due to occult derangement of the medial meniscus J Bone & Joint Surg 36-A 88 1954

Injuries of the Medial Meniscus

FREDERICK LEE LIEBOLT, M D *

Injuries to the medial meniscus occur in various ways for which many explanations are given. All authors agree that the most common force is the temporary fixation of the foot at the time that the knee is driven inward, but most reports state that the meniscus is trapped and crushed between the condyles. However the author believes that, as the femur rotates internally the femoral attachment of the tibial collateral ligament also is rotated internally and posteriorly, which in turn, because the meniscus is attached to the ligament, fixes and immobilizes the meniscus. Then if the tibia is in external rotation, undue stress is placed on the anterior attachment of the meniscus; if in internal rotation, undue stress is placed on the posterior attachment and if in neutral position, undue stress is placed on the peripheral attachment. Such forces initiate the injury but the degree of violence determines the type: a lesser force loosens stretches or tears the attachments, while a greater force splits the meniscus longitudinally or jerks it in two transversely.

The injuries may be of many types and not necessarily longitudinal, as commonly thought. Although longitudinal ruptures are the most common, there may be tears, which are transverse, oblique, horizontal or incomplete tabs, fringes, crushing or fragmentation, stretching or tearing of the attach-

ments as well as combinations of the above (Figs 1 to 3). In the author's first 111 consecutive cases the above types of injuries were present in 18 different positions, with longitudinal tears taking precedence over the next most common, transverse tears, in the ratio of 87 to 1.

The medial meniscus is injured more frequently than the lateral meniscus because, being attached to the tibial collateral ligament, it cannot rotate with the tibia as freely as the lateral meniscus which is unattached to the fibular collateral ligament. The author's statistics reveal tears of the medial meniscus to be 6 times more common than tears of the lateral meniscus. Other reports vary between 17 to 1 (Smillie¹), 26 to 1 (Dunn²), 37 to 1 (Dickson³), 51 to 1 (Bristow⁴) and 37 to 1 (McMurray⁵).

The sensation of weakness, slipping, instability or the actual giving way of the joint is the most common symptom of a torn meniscus and is much more common than locking. It is due to the torn portion of the cartilage moving under the pressure of the femoral condyle on the tibial condyle. Such knees are more prone to injury and each time the knee gives way or locks, pain, swelling and temporary disability occur. Each additional injury makes the knee more susceptible to further injury and produces minimal trauma which is accumulative and over a period of years produces osteoarthritic changes within the joint. Persons with torn menisci do not like to walk over rough terrain or down steps and are fearful

*Department of Surgery (Orthopedics) of the New York Hospital—Cornell Medical Center, New York. This study was aided by a grant from a charitable trust.

FIG 1 (Left)
Medial meniscus
showing minor
tear



FIG 2 (Right top)
Medial meniscus, showing
major tears.



FIG 3 (Right bottom)
Medial meniscus, showing
tear in an elderly person
aged 71



of any motion which produces internal rotation of the knee joint. Finally the patient develops a great fear of instability and seeks medical aid in the form of elastic bandages, braces, or surgery.

Thus the history obtained from the patient is of great diagnostic importance and from it the proper diagnosis can be made. No other feature of the work up is as important. Without a proper and detailed record of all past events, an accurate diagnosis is impossible. Frequently patients are seen during an intervening period when there are no positive physical findings and yet, from the history alone the diagnosis can be established.

The principal positive physical finding is tenderness localized to the medial joint line. Such tenderness must be differentiated from

tenderness of the tibial collateral ligament, and careful palpation is necessary to determine this. In fact, the areas of tenderness involving the ligament or the meniscus may approximate each other so closely that surgery may be dependent upon the width of one finger that is tenderness of the ligament indicates conservative treatment, while tenderness of the meniscus one finger's width away indicates operation. The frequency of negative findings in exploratory arthrotomies is due to this point, i.e., the failure to determine accurately before operation the principal and maximal site of tenderness.

Additional information is gained through 8 signs 7 by the author and 1 by McMurray.* The first 3 are related to the menisci directly and are based on the

phenomenon that the menisci are pressed between the femoral and the tibial condyles only upon hyperextension and hyperflexion. The next 4 differentiate between injuries of the menisci and the collateral ligaments. The 7 signs are (1) the passive snapping of the knee into hyperextension by the examiner which produces pain localized to the medial joint line for injuries to the medial meniscus, or to the lateral joint line for injuries to the lateral meniscus (2) the passive forcing of the knee into hyperflexion, which produces pain localized to the affected meniscus (3) the active snapping of the knee into hyperextension by the patient, which produces protective limitation of motion of the affected knee (4) the forceful abduction of the knee, which produces pain referred to the tibial collateral ligament and indicates a ligamentous rather than a cartilaginous injury, (5) the forceful adduction of the knee which produces pain referred to the fibular collateral ligament and indicates a ligamentous injury (6) the sharp internal rotation of the tibia by a quick twist of the ankle which produces sudden pain localized to the medial joint line as the tibial collateral ligament tugs on the injured meniscus and (7) the sharp external rotation of the tibia, which often produces sudden pain referred to the lateral joint line. This last sign is less specific because the fibular collateral ligament is not attached to and does not tug on the lateral meniscus. Should the last two signs produce pain at either of the collateral ligaments instead of the joint lines a ligamentous injury must be considered. The examiner should be cautioned to be ready to interpret the rotation signs at the time they are performed because they produce exquisite pain and the patient will not allow them to be repeated.

The eighth sign is that of McMurray.* It consists of an auditory or palpable click in the knee joint when the leg is rotated back and forth, while the knee is held in maximum flexion (heel to thigh) and again in 90° flexion (right angle). According to

McMurray this sign is useful only for injuries from the middle to the posterior attachment of either cartilage and "is of little value when the lesion is anterior to the mid-line of the joint."

The above signs are helpful but rarely are all of them present in the same patient, and in some instances of proved meniscus injury, none is positive. Therefore, although useful, they are not to be depended upon entirely for the diagnosis.

The term "locking" needs to be defined, since some reports state that locking occurs in practically every case of injury to the medial meniscus, while other reports state with equal emphasis that locking is rare. The difference rests upon the definition of the term. It is true that a 25° flexion deformity with inability to reach complete extension does exist in all acute injuries of the menisci, but such failure of extension is functional rather than mechanical and is due to pain spasm and effusion. True locking is a mechanical defect, in which the inner portion of a longitudinally torn cartilage shifts either to the mid-line of the condyles or beyond to the intercondylar fossa or a transversely torn cartilage is caught between the condyles anteriorly or a loose body is trapped between the condyles. Thus in such instances as the joint is closed in extension, the offending structure mechanically prevents the rolling together of the condyles much the same as a stick prevents the rolling together of the hinges upon closing a door.

Locking of the joint does not always occur; in fact, there are more torn cartilages which do not lock the knee joint than there are that do. Although locking may occur at the time of the original injury generally it becomes much more frequent long afterward when the inner fragment becomes more hypermobile and slips back and forth with ease across the tibial condyle. Over a period of time some patients learn to unlock the knee but in a first injury and in displacement of a longitudinal fragment beyond the mid-line of the joint it

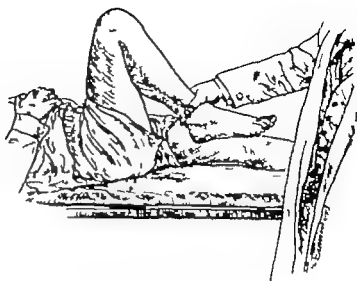
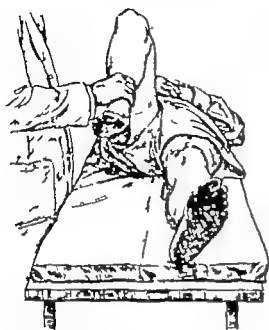
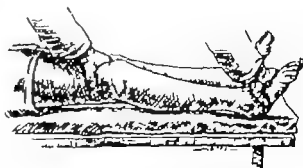
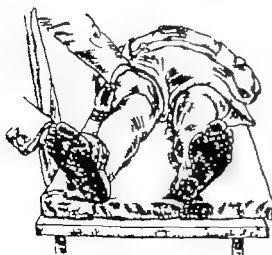


FIG 4 Author's technic for unlocking the knee joint (A) First maneuver demonstrated by front and side views, in which the calf is forced firmly against the thigh.



(B) Second maneuver demonstrated by front and side views, in which the femur is firmly rotated internally and the tibia is firmly rotated externally



(C) Third maneuver demonstrated by front and side views, in which the knee is extended slowly while the femur is held in internal rotation and the tibia in external rotation

necessary for the physician to employ reduction measures. The methods used vary with different surgeons and consist of manipulation under anesthesia: traction to the leg, snapping of the knee, kicking of the leg, shaking and rocking of the joint, abduction and adduction of the tibia, and open operation.

The author's technic for unlocking the knee joint has been reported previously⁷ (Fig. 4). Briefly, it is performed without anesthesia by placing the patient in a supine position and forcing the calf of the leg against the thigh. Then, with the femur held internally rotated and the tibia markedly rotated externally the knee is slowly extended. Should the manipulation be unsuccessful as demonstrated by the Jones sign in which the leg fails to extend to 180° surgery by open operation is indicated, rather than further manipulation under anesthesia.

In 1909 Sir Robert Jones⁸ established a conservative surgical viewpoint, which has been maintained to date when he stated, "I refuse to operate in any case I see early the subject of a first derangement. In order to follow the previous advice and to give the patient the benefit of the doubt in the hope that the first injury may not be a tear of the meniscus or if so that it may heal as some believe the patient is treated by aspiration, a roller elastic bandage, ice packs and rest, followed after 48 hours by heat, massage and ambulation. The author does not subscribe to the use of plaster casts because they are cumbersome, incapacitating, produce limitation of motion and have no bearing on the healing of a torn cartilage to date no satisfactory evidence has been offered that torn cartilages heal satisfactorily even peripherally at the site of their blood supply.

All patients will recover from the first injury whether or not the meniscus is torn. However, when instability, slipping, giving way, locking and recurrent injuries occur arthrotomy for removal of the medial meniscus is indicated. The procedure should

be performed under tourniquet control for visibility and for the prevention of added trauma by constant sponging.

For acute injuries, there is no need at any time to make long incisions or to expose the knee joint widely. Normal anatomic findings preclude the use of long incisions because the tibial condyle, the medial joint line and the infrapatellar branch of the saphenous nerve prevent the use of an incision below the joint line and the belly of the vastus medialis muscle limits the proximal extent of the incision. McMurray has reported two cases in which long incisions produced recurrent dislocation of the patella. Only in other conditions, such as knee fusion or synovectomy, are large incisions indicated or necessary.

Adequate exposure is gained by a well-placed longitudinal anteromedial incision extending proximally from the joint line for not more than 6 to 8 cm. Through such an incision the entire joint can be visualized except for the suprapatellar pouch, which is best investigated by the palpating finger. It is the only time, however, that it is necessary or proper to place a finger in the joint. Should physical findings or exploration reveal the necessity of approaching the lateral aspect of the joint, a similar incision is made anterolaterally. It must be remembered that one of the greatest factors in successful knee joint surgery is to prevent, and to keep to a minimum, surgical trauma to the joint. Bruising of the articular cartilages by knife cuts, instruments, excessive sponging and retractors is unforgivable. The incision, of course, should be adequate but such additional trauma as incising normal muscle tissue, cutting sensory nerves, dissecting the capsule from its normal attachments, incision into the suprapatellar pouch, sectioning ligaments and displacing the patella for exposure are all unnecessary and are to be condemned with emphasis.

After detailed exploration of the joint, the medial meniscus should be detached anteriorly, excised from the coronary and the tibial collateral ligaments and removed as

far posteriorly as possible through one incision. Forty years ago Sir Robert Jones advocated the removal only of the torn portion of the cartilage, but today this is considered to be an unsound surgical procedure. In three patients operated upon recently by the author complete relief of symptoms was obtained by removing peripheral portions of normal, not regenerated, meniscus left behind by other surgeons. However, this statement should not be interpreted to mean that it is necessary to remove the posterior portion of the meniscus as advocated by Bosworth⁹ and others, because there is adequate evidence on record that the proper removal of a cartilage anteriorly does not produce additional symptoms furthermore the author is acquainted with one case performed by an other surgeon in which the popliteal artery was injured, requiring amputation of the leg of a nurse.

Passive motion is started on the day after operation and active motion on the third day. The patient is allowed up the seventh or the eighth day after operation, wearing a 4-inch roller all-cotton elastic bandage and is discharged from the hospital without crutches or cane. The sutures are removed the tenth day after operation. The author's postoperative regimen for meniscectomy of the knee joint has been given in detail in another publication.¹⁰ A plaster cast after operation is not necessary and is definitely contraindicated because of the stiffness of the knee which follows. Casts plus the production of unnecessary trauma at operation have been the cause of the firm feeling and well-taken stand by laymen, as well as by many physicians, that operation upon the knee joint should not be performed because of the great danger of stiffness and immobility.

Much confusion exists as to the prognosis following removal of menisci. Physicians, laymen, athletes, compensation courts, athletic directors and army chiefs, frown upon the procedure. Almost every person knows of a case that has not turned out well. The general feeling is that once the knee is op-

erated upon the joint never again will be the same. There is adequate reason for this belief. Improper diagnosis, inadequate surgery, poor operative technic, infection and immobilization are the causes of poor results.

With the above factors eliminated however, there still remains the fact that many patients cannot return to full activity. Upon this question hinges the great difference of opinion. However the answer is simple. The error is made in grouping together all cases of meniscus injury without separating the pure meniscus injuries from those complicated by other injuries to the knee joint. For example if a torn anterior cruciate ligament exists in conjunction with a torn meniscus obviously the removal of the meniscus will not return the joint to normal. It is patently unfair to blame the disability from a torn ligament on the fact that the meniscus is removed or that the knee is operated upon. Without any of the above complications, the removal of a torn cartilage can be likened to the removal of a diseased appendix. The operation is as clean-cut, the convalescence is the same and the results as good that is a normal patient who can return to full activity.

REFERENCES

1. Smilie I. S. Injuries of the Knee Joint, Baltimore. Williams & Wilkins, 1951.
2. Dunn, N. Observations of some injuries of the knee joint, *Lancet* 1:1267 1934.
3. Dickson, F. D. Injuries of the knee joint, *J. A. M. A.* 110:122, 1938.
4. Bristow W. R. Internal derangement of the knee joint, *J. Bone & Joint Surg.* 17:605 1935.
5. ——— Internal derangement of the knee joint, *Am. J. Surg.* 43:458 1939.
6. McMurray T. P. The semilunar cartilages, *Brit. J. Surg.* 29:407 1942.
7. Liebolt, F. L. Tears of the medial meniscus, *S. Clin. North America* 30:555 1950.
8. Jones Robert. Notes on derangement of the knee, *Ann. Surg.* 50:969 1909.
9. Bosworth, D. M. Operation for meniscectomy of knee, *J. Bone & Joint Surg.* 10:1113 1937.
10. Liebolt, F. L. and Stein J. J. Physical

reconditioning of the knee following removal of menisci, Arch Phys. Med 27 413 1946

- 11 Brantigan, O. C. and Voshell A. F. The mechanics of the ligaments and menisci of

the knee joint, J Bone & Joint Surg 23 44 1941

12. King, Don. The healing of semilunar cartilages, J Bone & Joint Surg. 18:333 1936

Recurrent Dislocation of the Patella

DUNCAN C. MCKEEVER, M.D.

Recurrent dislocation of the patella includes only cases in which the patella is in normal position at birth and continues to occupy a fairly normal position throughout life. Because of developmental or congenital anatomic variations which alter the functional mechanics of the knee joint such patellae may dislocate momentarily or for longer periods under certain circumstances. Under extreme conditions, usually after several recurrences the dislocation may become fixed. This definition excludes those comparatively rare cases in which the patella is misplaced at birth and never occupies a normal position unless placed in this position surgically. The definition does not exclude all traumatic dislocations, which may under some conditions become recurrent. Trauma may cause dislocation of the patella in any knee but spontaneous recurrent dislocation does not ensue in normal knees.

Recurrent dislocation of the patella is essentially a mechanical problem. When it occurs the quadriceps muscle supplies the dislocating force. Dislocation is possible only when this force is applied in an abnormal direction because of developmental anatomic variations and faulty co-ordination.

One case of recurrent medial dislocation of the patella has been reported, following poliomyelitis,¹ but for all practical purposes recurrent dislocation of the patella is confined to lateral dislocations. Dislocation may be complete or merely a subluxation which produces momentary instability or mechanical derangement of the knee joint and re-

turns to normal position spontaneously and so quickly as to be difficult to recognize and diagnose correctly.

Dislocation of the patella can occur only when the medial line of transmission of the contractile force applied by the quadriceps and projected through the patella to the tibial tubercle is not a straight line but is angled medially with the apex of the angle at the center of the patella. Without this one factor dislocation cannot occur and this fact is the key to the successful treatment of the condition. Since the greatest strength of ligament and tendon is attached to the patella, the patella will tend to be displaced so that the force is transmitted in a straight line when a pull is applied to it by the muscle just as any curved bent or twisted object fastened at one end will tend to assume a straight line if tension is applied to the other end. The deviation of the patella from this straight line between the center of quadriceps pull and the tibial tubercle may be accentuated when the knee is slightly flexed and more particularly if there is external rotation of the knee in the flexed position. Therefore it is more probable that spontaneous dislocation will occur in this position, and probably it is impossible for it to occur if the knee is in complete extension except in the presence of extreme anatomic variation. Subluxation may occur in this position.

Several secondary factors contribute to the mechanics of recurrent dislocation. A poorly developed vastus internus, a high

- reconditioning of the knee following removal of menisci, Arch Phys. Med. 27 413 1946
- 11 Brantigan O C and Voshell, A. F The mechanics of the ligaments and menisci of the knee joint, J Bone & Joint Surg. 23 44 1941
- 12 King, Don The healing of semilunar cartilages, J Bone & Joint Surg. 18,333 1936

Recurrent Dislocation of the Patella

DUNCAN C. MCKEEVER, M D

Recurrent dislocation of the patella includes only cases in which the patella is in normal position at birth and continues to occupy a fairly normal position throughout life. Because of developmental or congenital anatomic variations which alter the functional mechanics of the knee joint such patellae may dislocate momentarily or for longer periods under certain circumstances. Under extreme conditions, usually after several recurrences, the dislocation may become fixed. This definition excludes those comparatively rare cases in which the patella is misplaced at birth and never occupies a normal position unless placed in this position surgically. The definition does not exclude all traumatic dislocations which may under some conditions become recurrent. Trauma may cause dislocation of the patella in any knee but spontaneous recurrent dislocation does not ensue in normal knees.

Recurrent dislocation of the patella is essentially a mechanical problem. When it occurs the quadriceps muscle supplies the dislocating force. Dislocation is possible only when this force is applied in an abnormal direction because of developmental anatomic variations and faulty co-ordination.

One case of recurrent medial dislocation of the patella has been reported, following poliomyelitis,¹ but for all practical purposes recurrent dislocation of the patella is confined to lateral dislocations. Dislocation may be complete or merely a subluxation which produces momentary instability or mechanical derangement of the knee joint and re-

turns to normal position spontaneously and so quickly as to be difficult to recognize and diagnose correctly.

Dislocation of the patella can occur only when the medial line of transmission of the contractile force applied by the quadriceps and projected through the patella to the tibial tubercle is not a straight line but is angled medially with the apex of the angle at the center of the patella. Without this one factor dislocation cannot occur and this fact is the key to the successful treatment of the condition. Since the greatest strength of ligament and tendon is attached to the patella, the patella will tend to be displaced so that the force is transmitted in a straight line when a pull is applied to it by the muscle just as any curved bent or twisted object fastened at one end will tend to assume a straight line if tension is applied to the other end. The deviation of the patella from this straight line between the center of quadriceps pull and the tibial tubercle may be accentuated when the knee is slightly flexed and more particularly if there is external rotation of the knee in the flexed position. Therefore it is more probable that spontaneous dislocation will occur in this position, and probably it is impossible for it to occur if the knee is in complete extension, except in the presence of extreme anatomic variation. Subluxation may occur in this position.

Several secondary factors contribute to the mechanics of recurrent dislocation. A poorly developed vastus internus, a

placement of the patella, or a poorly developed lateral condyle may singularly or in combination, be definite factors in recurrent dislocation of the patella. Usually all three are present. These anatomic abnormalities as entities cannot be congenital; they are developmental end results of the effect of altered function on the rate and the direction of growth, or the effects of physiologic variation on the quality of growing tissue. No one of them acting alone can cause recurrent dislocation of the patella.

If the vastus internus never plays a normal part in extension of the knee it never develops to its proper volume and strength. This is a functional lack of development and is the corollary of functional hypertrophy which occurs when a muscle is called upon to do more than its normal share of work. Each is a physiologic response and neither is indicative of a defect in the muscle itself.

In normal anatomic position the lower

pole of the patella is generally considered to be level with the knee joint, and an abnormally high patella is an essential anatomic variation in recurrent dislocation (Fig. 1). If the patellar tendon is of normal length dislocation of the patella is impossible, except in case of complete or partial traumatic avulsion of the insertion of the tendon. On the other hand, there are many patients in whom the patella may be 1 inch or even $1\frac{1}{2}$ inches above the normal position who never develop dislocation of the patella because the direction of pull of the quadriceps, projected through the patella to the tibial tubercle is a straight line.

The patella is invariably at a higher position than normal in boys who have had Osgood-Schlatter disease. This condition must alter the quality of the tissue in the growing tendon at its insertion. The faulty maturity rate of newly formed bone in the epiphysis at the tubercle must be associated

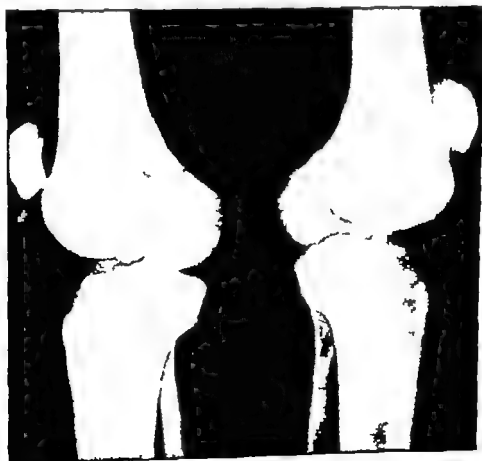


FIG. 1 High patella—an essential factor in recurrent dislocation.

with a faulty maturity rate in the connective tissue of the growing tendon or an abnormal relative amount of elastic fiber in the tendon

Very high placement of the patella is usual in cases of chondromalacia of the patella of nontraumatic origin and associated with this, an extensive plica synovialis or perhaps a completely closed suprapatellar pouch is a common finding. This is a clinical observation and the relation of these entities has not been studied. It may be a hereditary variant, or the two conditions may be coincidental but not directly related. It seems reasonable to assume that the patellar cartilage, forming and functioning in this abnormally elevated position without constant contact with an opposed cartilaginous surface is of inferior quality and not normally resistant to the trauma incident to passing over the articular margin of the femur into the intercondylar groove.

The relative size of the medial and the lateral condyles of the femur in the adult is determined by the effects of function on growth. The greatest growth occurs at the point of greatest function. The chief function of the lower end of the femur is to transmit stress in weight bearing. If this stress line starts with the normal contact of a normal foot on the ground it will pass through the physiologic centers of the lower tibial, upper tibial and lower femoral epiphyses in both planes. Constantly maintained normal direction of growth will result in a straight leg in the adult and normal relative size in the femoral condyles. If the foot is not normal anatomically or if the stress lines are displaced otherwise deviation in the position of these stress lines with relation to the physiologic center of the growing epiphyses will produce an alteration in the direction and the relative rate of growth. Developmental abnormalities will occur in proportion to the degree of the defects in the feet, modified by the static and dynamic resistance to these defects. The effects of these forces have been discussed elsewhere.² In the application to the prob-

lem of the dislocation of the patella, if the weight bearing stress lines pass medial and posterior to the physiologic center of the lower femoral epiphysis growth in the medial condyle is accelerated, and growth in the lateral condyle is retarded, consequently this condyle never gets to be as large as it would be if it functioned normally. The overall deformity in the leg resulting from medial and posterior displacement of the weight-bearing stress lines at the knee is a genu valgum with external torsion of the tibia, a condition found in almost every case of recurrent dislocation of the patella. If in such a case the patella is also high, the absence of a normal amount of pressure by the patella against the anterior surface of the lateral condyle will result in a lessened growth rate in this area also. The anterior projection of the lateral condyle will be lessened, and the intercondylar groove will be much more shallow than it would grow to be in the presence of a normally positioned and normally functioning patella.

Given then (1) a knee in which the patella is placed so high that under tension, with the knee in extension it does not rest within the intercondylar groove but must enter into it when flexion takes place (2) a small lateral condyle to either side of which the patella may pass with almost equal facility (3) a knee in which there is medial angulation of the line of force transmitted by the quadriceps through the patella to the tibial tubercle dislocation becomes a possibility in direct proportion to the degree of these various defects and a certainty if the defects are of sufficient magnitude and the parts played by each factor are activated concurrently.

TREATMENT

There is one anatomic condition without which no spontaneous dislocation of the patella can occur. This is the medial angulation in the line of force transmitted by the quadriceps through the patella to the tibial tubercle. It follows therefore that if this angulation is removed and the force is trans-

mitted through the patella to the tibial tubercle in a straight line dislocation cannot occur. There are two methods by which this can be brought about.

1 Osteotomy of the lower end of the femur with correction of the genu valgum will straighten out this line of force and stop recurrent dislocation but may produce an undesirable alteration in the functional axis and plane of knee-joint motion. Osteotomy entails a rather long disability. It does not lower the level of the patella with relation to the knee joint but may even increase this defect. Such elevation, if uncorrected, will in almost every case regardless of cause, eventually lead to chondromalacia of the patella and chronic synovitis. On the basis of long-range results osteotomy is not the best procedure.

2 The other method of bringing the line of force from the quadriceps through the patella to the tibial tubercle into a straight line is to move the insertion of the patellar tendon. Transposition of half of the tendon medially may be sufficient to prevent dislocation in mild cases but it does not significantly lower the patella, and the partial medial displacement of the functional center of the tendon insertion is not sufficient in a great many cases to prevent dislocation. Simple medial displacement of the tibial tubercle will prevent dislocation but it does not lower the level of the patella and therefore is not the most satisfactory operation to be carried out.

The best functional result is obtained when medial transposition of the tibial tubercle is combined with distal transposition sufficient to lower the patella to normal position. Variations of transplantation of the tibial tubercle have been described by a number of authors being mentioned by Mouchet and Durand in 1921 and by others later.^{3,4,5} Chief variations in these operations seem to lie in whether or not the tibial tubercle was advanced distally as well as medially and in the method of holding it in place after its transposition. Various means employing screws and plates, or screws and

plates combined with countersinking, have been described, and both Smillie and the author have described methods of transplanting the tibial tubercle in such a manner that it becomes self locking without the use of a screw or a plate.^{4,5} Either of these methods will fix the transplanted tibial tubercle securely in place and permit very early resumption of unprotected function. The authors' operation is carried out as follows.

Through a median parapatellar incision extended well below the tibial tubercle and just above the patella, the patellar tendon is exposed down to its insertion. The lateral expansions of the extensor mechanism are incised in a linear direction along the edge of the patellar tendon and the patella, to a point near the upper border of the patella. The tibial tubercle is cut out in a block about $\frac{3}{8}$ by $\frac{1}{2}$ inches the proximal end of which is vertical, the distal end of which is undercut obliquely at an angle of 45° so that the block can be removed only by elevating the proximal end and slipping the distal end out from beneath the overhanging edge of the tibia. Then traction is made on the tendon until the lower pole of the patella is level with the knee joint. The tibial tubercle is carried medially until the patella lies directly over the intercondylar groove. This position is marked, the periosteum is removed, and a block of bone is cut out, with the distal edge undercut in similar fashion to leave a hole the exact size and shape of the tibial tubercle block. The tibial tubercle with the attached patellar tendon is then inserted into this new site by slipping the distal end in first, then pushing or driving in the proximal end. No amount of traction in the direction of pull of the quadriceps muscle can displace it. The block of bone removed from the new insertion is put into the site of the old insertion in order to fill this defect in part. The capsule and the lateral expansions of the quadriceps mechanism are closed as well as possible. This is ordinarily quite easy on the medial side and difficult, or perhaps im-

possible, on the lateral side, but if a defect persists at this point it does not affect the end result. After closure, a pressure dressing is the only immobilization necessary. This dressing can be removed in 4 or 5 days to begin active motion. It is possible to start full weight bearing within a week and 90° of flexion should be acquired within 3 to 6 weeks. A full range of motion should be attained eventually (Fig. 2).

This operation does not solve the problem of recurrent dislocation before epiphyseal closure. If tibial tubercle transplant is done before epiphyseal closure deformity will result in proportion to the remaining growth potential in this epiphysis. If recurrent dislocation is a problem during the growing period, efforts should be made to treat the condition conservatively. Alterations of weight bearing alignment may be used in young children to restore direct

quadriceps pull through the corrective effect of normal function on the direction of growth. Operative procedure on the soft parts such as transfer of part of the tendon to a point medial to the tibial tubercle may be used. Ligamentous slings may be employed as a temporary measure. Tenotomy and lengthening of the vastus lateralis component of the quadriceps might be effective in some cases. All these procedures might be temporary expedients until the definitive and permanent procedure described above could be carried out unless correction of alignment is possible by the conservative alteration of direction of growth by altered function or by stapling. This would constitute the only exception.

Patellectomy as a solution to the problem of recurrent dislocation of the patella is mentioned only to be condemned. Obviously if the patella has been removed it



FIG. 2. Level of patella before and after operation

cannot dislocate, but the tendon can and it may become chronically dislocated. Patellectomy leaves the lower end of the femur without protection in kneeling and in falls. Calcification within the tendon is a common and very disabling late end result. If chondromalacia of the patella is so far advanced that a painful joint, with further traumatic arthritic changes in the condylar articular surfaces is to be anticipated, further distal displacement of the patella, so that it lies over the fat pad or replacement of the patellar articular surface with a prosthesis produces a much more satisfactory result than patellectomy.

SUMMARY

Recurrent dislocation of the patella is a mechanical problem brought on by a com-

bination of developmental variations in functional anatomy of the knee joint. The operation of choice is medial and distal transposition of the tibial tubercle.

REFERENCES

- 1 Zadek, Isadore. Recurrent medial dislocation of patella, *Bull. Hosp. Joint Dis.* 8: 30, 1947.
- 2 McKeever, D. C. Interpretation and treatment of weight-bearing defects in apparently healthy children, in *Clinical Orthopaedics* 1: 56, 1953.
- 3 Thompson, F., and Bosworth, D. M. *Am. J. Surg.* 73: 335-9.
- 4 Smillie, I. S. *Injuries of the knee joint*, Baltimore, Williams & Wilkins, 1951.
- 5 McKeever, D. C. Transplantation of the tibial tubercle, *J. Bone & Joint Surg.* 33: 478, 1951.

Treatment of Dislocation of the Patella

FRITZ TEAL, M D *

Dislocation of the patella may be either simple or chronic and recurrent. It occurs twice as frequently in females as in males and tends to be familial.

ETIOLOGY

The etiologic or causative factors are varied and include

- 1 Congenital (including agenesis of the patella)
- 2 Valgus of the knee (including the natural valgus of the female knee)
- 3 Relaxation of internal lateral ligament of knee
- 4 Deformity of the outer femoral condyle either congenital or from partial arrest of the lower femoral condyle outer portion
- 5 Traumatic i.e. external violence as in football in this case the internal collateral ligament may be damaged.

MECHANISM OF DISLOCATION OF PATELLA

The mechanism of dislocation of the patella is similar in all etiologic groups. The knee is partially flexed the lower leg and the foot are rotated externally and abducted below the knee. This is true of all cases including those in which external violence or trauma is a factor as in football injuries. Therefore attention is called here to the fact that many injured knees have been operated upon for internal derangement where the true pathology is patellar dislocation.

COMPLICATIONS

Complications of dislocation of the patella are mainly chondromalacia of the patella and eventual degenerative knee joint changes. Quadriceps atrophy also occurs and is a factor in recurrent dislocation.

TREATMENT OF SIMPLE PATELLAR DISLOCATION

The treatment of simple or first dislocation is important in the prevention of recurrences, as it is in cases of shoulder dislocation. In most instances there is an early hemarthrosis or joint effusion which should be relieved by aspiration. Ice packs relieve pain and reduce subsequent hemorrhage and further effusion. The most important factor in preventing recurrences is immobilization of the knee joint. This is accomplished best by the use of a circular cast applied over moleskin strap turned back at the bottom or over stockinet which has been liberally soaked with Ace adherent. The knee should be in full extension so that quadriceps setting can be started immediately and weight-bearing allowed as soon as the knee is pain free.

After removal of the cast in 6 weeks physical therapy is essential to aid in restoring joint motion and the quadriceps mechanism. The latter is important in preventing recurrences. A convalescent support (knee cage brace which limits flexion to 90°) is used for at least 6 weeks. In addition the natural valgus strain of the knee is partially eliminated by elevation of the inner side of the

* Lincoln, Neb.

heel and the use of a scaphoid pad. However, if patellar dislocation recurs in spite of the aforementioned treatment, surgery must be considered.

SURGICAL TREATMENT

Surgical treatment of recurrent dislocation of the patella should have the following criteria as its goal

- 1 Prevention of recurrences
- 2 Prevention or alleviation of complicating chondromalacia of patella, thus indirectly militating against general chronic joint degenerative changes
- 3 Restoration of quadriceps mechanism without loss of knee stability or mobility
- 4 All of the above without producing painful joint.

The surgical treatment of recurrent dislocation of the patella is almost as varied as that for bunions. Many of the operations are modifications of the Goldthwait operation which I reported on 20 years ago. These operations all tend to check lateral displacement of the patella by transplantation of the outer one half of either the patellar ligament or the quadriceps tendon to the medial side of the joint (Fig. 2). As Dr. H. W. Orr points out in a recent history of surgical procedures for patellar dislocation, Dr. Joel Goldthwait also transplanted the entire patellar insertion medially in 1900 thus preceding by many years the modern exponents of this method.

Surgical procedures for the treatment of recurrent dislocation of the patella fall roughly into 4 categories

- 1 Incision of the lateral capsule and either incision and plication of the medial capsule or excision of an elliptical portion of the medial capsule with displacement of the patella medially for example the Krogius operation (Fig. 1)
- 2 Fascial transplant, with or without medial capsular excision or shortening, for example Ober Campbell, Gallie (Fig. 4)
- 3 Transplantation of all or part of the patellar ligament or its insertion medially sometimes combined with medial capsular

excision or plication for example Goldthwait and Hauser (Fig. 3)

4 Bone procedures such as osteotomy for the correction of valgus of the knee or elevation of the outer condyle of the femur by bone graft operation to deepen the intercondylar groove for example Albee (Fig. 5)

Experience has shown that the Krogius operation alone does not correct recurrent dislocation unless it is combined with some other procedure such as transplantation of the outer half of the patellar ligament or better still, transposition of the insertion of the patellar ligament medially. The fascial transplant, such as the Campbell, Ober Gallie or Soutter operations do not alone seem to be effective and are very apt to interfere with the quadriceps mechanism. Elevation of the outer femoral condyle by bone graft or other types of procedures alone as shown by experience seems to be inadequate. If this procedure is combined with transposition of the patellar insertion, good results may be obtained. Up to the present time, best results seemed to have been obtained with a combination of the Krogius procedure, with medial displacement of the patella and transposition of the patellar attachment medially. The big criticism lies in the fact that none of these operations can be effective or efficient if chondromalacia of the patella has already occurred. These operations or combinations of operations if done before the inferior surface of the patella is damaged, can give very excellent results. Unfortunately many of these patients seek medical aid after there has been a well-established degenerative disease of the articular surface of the patella. To this group of patients which forms the largest single group of recurrent dislocation of the patella, I have advised patellectomy.

AUTHOR'S METHOD

Several years ago excision of the patella was tried for recurrent dislocation of the patella with surprising success. By this method both chondromalacia and recurrent dislocation were eliminated, quadriceps mechanism

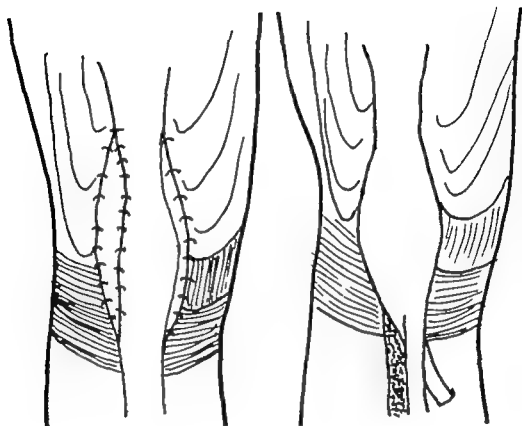
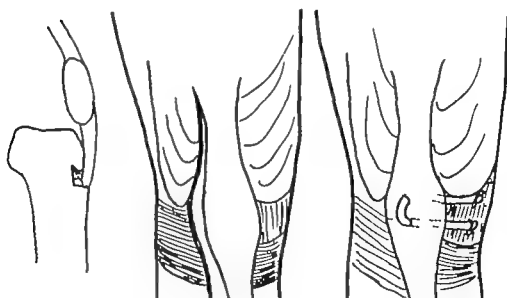


FIG. 1 (*Left*) Transplantation of elliptical piece of medial capsule to outer parapatellar incision. Closure of gap displaces patella medially (Kroggius⁶)

FIG. 2 (*Right*) Shifting outer one half of patellar ligament medially (Goldthwait⁴)



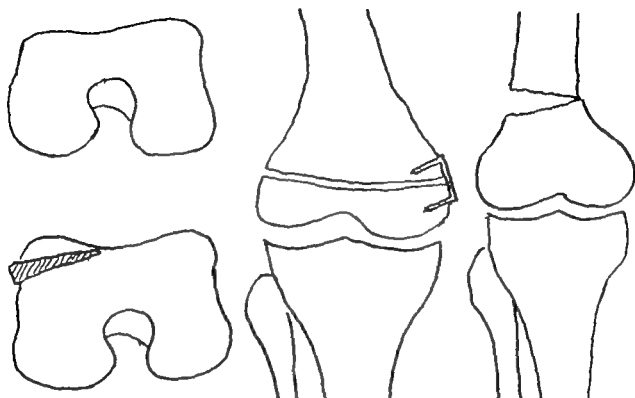


FIG. 5 (Left) Elevation of low outer condyle by bone wedge (Albee's) (Center) Stapling medial condyle to correct valgus (Blount) (Right) Osteotomy of femur to correct valgus

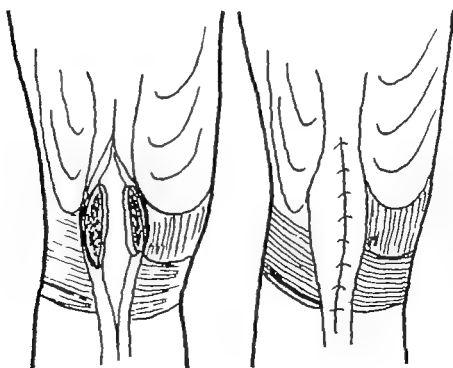


FIG. 6 (Left) Splitting patella for its excision (Right) Plication of patellar ligament and its expansions (Author)

was fully restored the knee moved through a normal range without pain. Since then 37 cases have been operated upon with excellent results, including 3 cases of bilateral recurrent dislocation. In 2 cases postoperative snapping of the outer portion of the quadriceps expansion occurred. After excision of an osteoarthritic ridge from the outer condyle the snapping ceased.

In this series best results have been obtained by splitting the patella and plicating the quadriceps ligament and its expansions after removal of the patella (Fig. 6). Fixation in a walking circular cast for 3 weeks is followed by an active physical therapy program. In one case manipulation of the knee was necessary because of difficulty in obtaining knee flexion. This patient had marked joint degenerative changes.

Experience has shown that excision of the patella for recurrent dislocation approaches fulfillment of the surgical criteria more than any other method. If the operation can be done before gross joint degenerative changes

have occurred the result is almost normal function. Excision of the patella does not destroy the quadriceps mechanism but does cure recurrent dislocation of the patella.

BIBLIOGRAPHY

- 1 Albee, F. H. The bone graft wedge in the treatment of habitual dislocation of the patella, *M. Rec.* 88:257 1915
- 2 Campbell, W. C. *Operative Orthopedics*, St. Louis, Mosby p. 451 1939
- 3 Gallie, W. E. and LeMesurier, A. B. Habitual dislocation of the patella, *J. Bone & Joint Surg.* 6:575 1924
- 4 Goldthwait, J. E. Slipping or recurrent dislocation of the patella, *Am. J. Orthop. Surg.* 1:293 1904
- 5 Hauser, Emil. Total tendon transplant for slipping patella. *Surg. Gynec. & Obst.* 66:199 1938
- 6 Krogius, A. Zur Operation Behandlung der habituellen Luxation der Knie Scheibe. *Zentralbl. Chir.*, March 5 1904
- 7 Ober, F. R. Tendon transplantation in lower extremity. *J. Bone & Joint Surg.* 17:774 1935

Avulsion of the Hamstring Tendons from the Ischial Tuberosity Report of Case

LEWIS M OVERTON M D * and

ROBERT ENGLAND, M.D *

The pathology the mechanism and the frequency of ruptures of the muscles and the tendons have been described or enumerated by a number of authors Gilcreest¹ enumerated the factors predisposing muscle and tendon rupture Brickner and Milch² were the first to list all conditions under which such ruptures may occur basing these on the observations made by Madyl.³

Certain muscles and tendons are more susceptible to rupture than others Conwell and Alldredge⁴ have listed the order of frequency as follows (1) muscles of the calf (2) extensors of the thigh, (3) biceps brachii (4) Achilles tendon, (5) long extensors of the thumb (6) supraspinatus (7) rectus abdominalis (8) extensors of the finger (9) adductor of the thigh and (10) triceps brachii. They did not mention rupture of the hamstrings

The rarity of the rupture of the hamstring muscles or tendons is indicated by only 5 case reports being found in the literature Janson⁵ in 1813 reported a case that had been seen by Roerder This was a young girl who had drowned and at autopsy was found to have a rupture of all of the hamstrings In 1817 Sedillot⁶ reported a rupture of both biceps femori No other cases were reported until 1903 when Ruedinger⁷

published a case of rupture of the biceps femoris from its attachment to the fibula. Edwards⁸ in 1932 presented a case in which the belly of the semimembranosus had been ruptured in a 24-year-old man while playing rugby football. Gilcreest⁹ reported a case of partial rupture of the belly of the semitendinosus in a 59 year-old man who received a direct blow over the muscle The case we wish to report is that of a complete avulsion of all tendons of the hamstrings from their attachment to the ischial tuberosity

CASE REPORT

A 55-year-old white man was admitted to the clinic on May 17 1952, with a complaint of being unable to run because of discomfort and insecurity of the right lower extremity

On February 25 1952, the patient slipped and fell while playing tennis forcibly flexing the right hip with the knee extended The fall was accompanied by a sudden momentary pain and a sound like a pistol shot in the right buttock area. This was followed by soreness, swelling and ecchymosis in the back of the upper right thigh The patient was able to walk with very little discomfort after one week The extremity remained insecure though it did not collapse, and he had been unable to run at all This gave him considerable concern because he always

had been extremely active and played tennis several times weekly

On examination at the time of admission there was a round, firm mass, the size of a lemon, about 5 inches below the right ischial

tuberosity. This lay beneath the deep fascia, was only slightly tender and was not fixed to surrounding tissues. It did move distally on active flexion of the knee but did not change position on hip motion.

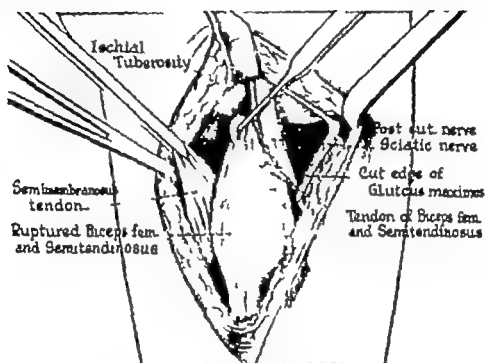


FIG. 1 Detached tendons and contracted muscle mass of the biceps femoris and the semitendinosus.

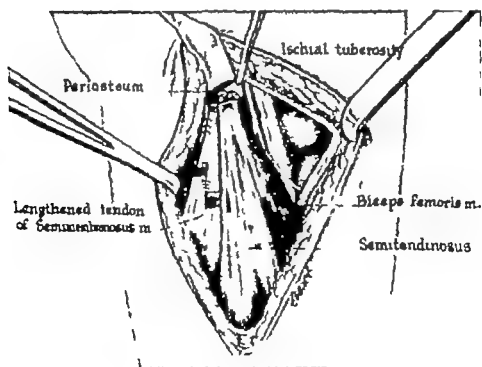


FIG. 2 Lengthening of the semimembranosus tendon and reattachment of all of the hamstrings to the ischial tuberosity

A tentative diagnosis of a rupture of the hamstring tendons was made however since the patient was in the fifth decade of life and could walk without difficulty we advised conservative treatment. This consisted of daily exercises. This was continued for approximately 2½ months. During this period the patient felt that there had been very little improvement. He had not regained either co-ordination or increase in strength. He wanted to become more active and return to playing tennis. For this reason surgery was advised and performed. At operation both the common tendon of the long head of the biceps femoris and semitendinosus and the tendon of the semimembranosus were completely detached from the ischial tuberosity. They were displaced distally for approximately 8 cm. the upper ends were doubled on themselves and were adherent to the sciatic nerve as it passed anteriorly (Fig 1). The loose ends of the tendons and the adjacent muscles were dissected free from the surrounding tissues. The tendons of the biceps femoris and the semitendinosus could then be approximated to the ischial tuberosity without difficulty. The semimembranosus was contracted markedly. This made it necessary to lengthen the upper tendinous portion of this muscle (Fig 2). The ischial tuberosity was then denuded of all soft tissues and roughened. A drill was passed about ½ inch from its tip. Then the tendons were reattached to the bone by heavy silk sutures. These were reinforced by suturing the periosteum to the tendons. The semimembranosus was further reinforced by suturing it to the tendon of the adductor magnus at its attachment to the ischium.

The wound was closed without drainage and a long leg plaster-of-Paris cast was applied with the knee in 90° of flexion. The patient was allowed up on crutches after 2 weeks. The cast was worn for 4 weeks. Mild

active contraction of the muscles was begun at this time. The active use of the muscles gradually returned. After 2 months there was good power but the patient had not completely regained co-ordination of the leg. He returned to playing tennis 6 months after surgery with practically the same agility that he had before the injury.

COMMENT

Rupture of the hamstring muscles and tendons follows the pattern of ruptures involving other muscles and tendons, as revealed by a review of the literature and the one case report. The rarity of rupture involving this group emphasizes that sudden forcible stretching of a muscle while it is in a state of active contraction is a most important etiologic factor. A survey of body mechanics will reveal that this can occur in the hamstrings only when the entire extremity is in extension with the torso being thrust forward forcibly. Such a condition is extremely rare. It is our belief that this explains the rarity of rupture in the hamstring group of muscles.

REFERENCES

1. Gilcreest, E. L. *J A M A.* 84:1819 1925
2. Brickner W M., and Milch, H. *Internat. Clinics* 2:95 1928
3. Madyt, K. *Deutsche Zisch. Chirurg.* 17:306 1882.
4. Conwell, H. Earle, and Alldredge, R. H. *Am. J Surg.* 35:36,22 33 1937
5. Gilcreest, E. L. *J A M A.* 100:153 1933
6. Edwards, H. C. *Lancet* 1:65 1932.
7. Riedinger J. *Munch m W Schr.* 34:1486 1903
8. Sedillot. *Memoire sur la rupture musculaire. Mém. et prix de la Société de méd. de Paris,* p 72, 173 1817
9. Janson L. *Essai sur les ruptures des tissus et des organes du corps humain.* Dissertation No 105 Université de Paris, 1813

10

Bicipital Tenosynovitis

ANTHONY F. DePALMA, M.D., AND
GERALD E. CALLERY, M.D.

It is most gratifying to the authors that an ever-increasing number of orthopaedic surgeons are becoming cognizant of bicipital tenosynovitis as a causative agent of pain and stiffness in the shoulder joint. Nevertheless, many workers still are reluctant to recognize the entity and to give it the place of importance that it deserves. Critical analysis of several hundred cases of painful shoulders encountered by the senior author in the past 7 years leads him to believe that tenosynovitis of the long head of the biceps brachii muscle is the most common antecedent of painful and stiff shoulders. This is true during both the younger and the older age periods of life. That implication of the biceps tendon may produce a painful shoulder is not a recent observation; this conclusion was recorded by many workers yet their teachings have not taken root, and the lesson remained to be relearned. Even Codman, who without a doubt contributed more to the comprehension of shoulder lesions than any worker in the past or the present, was of the opinion that the biceps tendon and its sheath played only a minor role if any in the production of shoulder symptoms. He wrote: "Personally I believe that the sheath of the biceps tendon is less apt to be involved than are the other structures. I have never proved its involvement in a single case. I think that the substance of the tendon of the supraspinatus is the most often involved."

Certainly Meyer must have recognized the significance of the biceps tendon in shoulder

lesions when he repeatedly recorded the alterations of this structure which he noted in the shoulders of cadavers (1921, 1926, 1928 and 1930). He masterfully described the degenerative and attritional abnormalities which he observed; these comprised fraying, shredding, fasciculations and tearing of the fibers of the biceps tendon. Some of his specimens exhibited partial or complete dislocation of the tendon out of the bicipital groove. In several shoulder joints the intracapsular portion of the tendon was absent, while the proximal end of the extracapsular portion had attained a bony anchor in the region of the lesser tuberosity. He proposed that the aforementioned alterations were the results of using the arm in a position of abduction and external rotation. Unfortunately this work was done on cadavers; hence the clinical significance of these findings could not be appraised conclusively. The authors have confirmed the work of Meyer both in cadavers and in living subjects and have recorded the findings and their clinical significance.

Stimulated by the work of Meyer in 1932, Pasteur attempted to link the alterations in the biceps tendon, as described by Meyer with the clinical picture characterized by pain and stiffness in the shoulder joint. He coined the term "teno-bursitis" to designate a new clinical syndrome in which involvement of the biceps tendon was believed to be the responsible etiologic element in periarthralgias and ankylosis of the shoulder joint. He was the first to incriminate the



FIG. 4 Marked hypertrophy of all the intracapsular structures except the labrum in the region of the comma's head also advanced fraying, shredding and hypertrophy of the biceps tendon. Note the advanced degenerative lesions in the musculotendinous cuff of this specimen.

FIG. 5 A specimen with complete avulsion of the musculotendinous cuff, advanced degenerative changes and hypertrophy of the biceps tendon. (DePalma, A. F. *Surgery of the Shoulder* Philadelphia, Lippincott)

generative changes which occur consistently in the biceps tendon after the third decade of life. What man or woman past 30 years of age has not at sometime or other complained of pain in the anteromedial aspect of the arm after a bout of excessive usage? Yet roentgenograms of the region fail to disclose any abnormalities. Once the process is precipitated all movements that require long excursions of the head of the humerus on the biceps tendon accentuate the pain. The movements most likely to elicit sharp twinges of pain are abduction and external rotation as well as internal and backward flexion of the arm. Some observers are of the opinion that in all cases of bicapital tenosynovitis degenerative abnormalities in the tendon are the responsible factors for the pathologic process. This is an erroneous

concept of this entity. It has been recorded previously that the entity has been encountered in individuals in the second decade of life (3 patients were 16 and 2 were 20 years of age) a period when degenerative alterations are absent or only minimal in severity. However it must be admitted that the increase in frequency of the lesion, during the decades of life when degenerative changes are known to exist in moderate and severe gradients indicates that such alterations enhance the precipitation of the inflammatory process and render the tendon-tendon sheath mechanism vulnerable to even minor traumas. In the younger age group anomalies of the bicapital groove, together with repeated trauma are the major factors in initiating the syndrome.

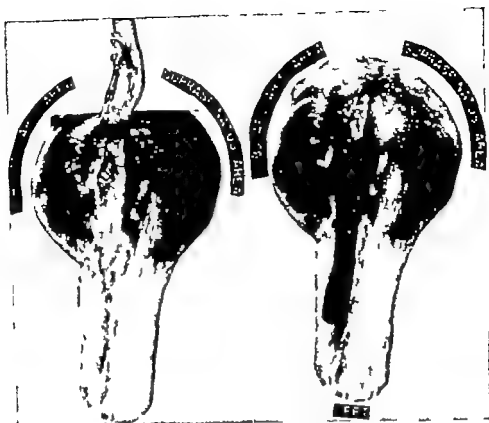


FIG. 6 (Left) Note defect in frayed biceps tendon and spur in the floor of the bicipital groove. (Right) When the tendon is in its normal position the defect in its substance fits snugly around the bony spur (DePalma A. F. Surgery of the Shoulder Philadelphia Lippincott)

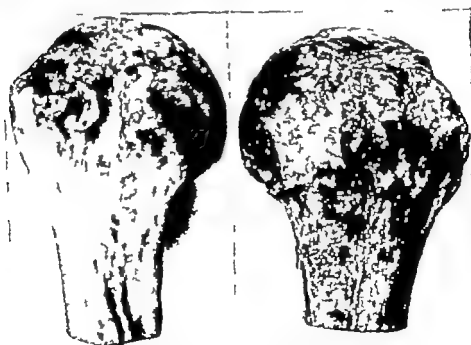


FIG. 7 (Left) Well-formed bony excrescences over both tuberosities. The intertubular sulcus is narrowed, and the head of the humerus proximal to the sulcus is flattened and eburnated. The biceps tendon of this specimen was thickened, frayed and shredded. (Right) Note complete atrophy and recession of tuberosities, obliteration of intertubular sulcus, excrescences in bicipital groove, and eburnation of the head of the humerus, anterior superior and posterior to the bicipital groove. The biceps tendon was thinned, frayed and shredded. (DePalma A. F. Surgery of the Shoulder Philadelphia, Lippincott)

ANOMALIES OF THE BICIPITAL GROOVE

Meyer was the first to point out developmental variations in the bicipital groove. He described a supratubercular prominence which is a ridge of bone continuous with the medial wall of the groove, extending proximally toward the articular head of the humerus. Its size and configuration vary and the type and the degrees of variations noted in a study of humeri, were recorded by Hitchcock and Bechtol. The senior author studied 100 humeri and noted that the angle of the medial wall of the bicipital groove was 90° in 23.9 per cent, 75° in 27.09 per cent, 60° in 21.7 per cent, 45° in 17.3 per cent, 30° in 7.3 per cent and 15° in 2.1 per cent (Fig 3). It was noted further that the supratubercular ridge was a pronounced structure in 23.8 per cent, moderately developed in 31.5 per cent and absent in 43.4 per cent. In addition, it was observed that the base of the supratubercular ridge was a factor in diminishing the depth

of the groove. This abnormality was noted in 9 per cent of the humeri with a supratubercular ridge. In these specimens the base of the ridge was unusually broad, continuing across the floor of the groove toward the lateral wall. In the light of these observations it is reasonable to conclude that such alterations force the tendon to function at a definite mechanical disadvantage. This is especially true of the shallow grooves and in cases with a supratubercular ridge. Both of these changes tend to force the tendon out of the groove against the transverse humeral ligament and are responsible for inflicting repeated traumas to the tendon and its gliding mechanism during movements of the arm. Under these circumstances the tendon and its sheath become even more vulnerable when severe strenuous exertions are superimposed on normal joint function, such as occurs in baseball tennis or laborious occupations. Also these abnormalities are agents in producing severe degenerative changes in the biceps tendon encountered after the fourth decennium.



FIG 8 (Left) Posterior aspect. Complete atrophy of the greater tuberosity and new bone formation on the posterior aspect of the shaft of the humerus below the level of the neck. (Right) Anterior aspect. Bicipital groove is completely obliterated by new bone formation. In this specimen no intracapsular portion of the biceps tendon was found. The proximal end of the extracapsular portion was buried in the osseous tissue obliterating the groove (De Palma, A. F. *Surgery of the Shoulder* Philadelphia Lippincott)

DEGENERATIVE PROCESSES

Physiologic wear and tear together with the aforementioned factors are responsible for degenerative changes in the tendon and alterations in the bicipital groove which increase in severity during each successive decade. The alterations comprise fraying, shredding and fasciculations of the tendon together with the formation of bony excrescences within the groove (Figs 4 5 6). In a previously recorded study it was interesting to note the severity of the lesions of the bicipital grooves and the tuberosities after the fifth decade. Irregular new bone formation in varying degrees over the tips of the tuberosities was an outstanding pathologic alteration (Fig 7). These altered the configuration of the bicipital sulcus rendering it narrow and in some instances obliterating the sulcus. Frequently bony spurs were encountered on the inner and the outer surfaces of the sulcus and within the floor of the groove. In the face of such changes it becomes obvious that they constitute a serious impediment to the normal excursion of the head of the humerus on the biceps tendon. Spur formations render the tendon vulnerable to repeated trauma and attritional alterations. These facts are supported by the severity of the degenerative changes noted in the biceps tendon.

After the fifth decade many humeral heads disclosed marked atrophy and recession of the tuberosities, and occasionally the intertubercular portion of the bicipital sulcus is worn away completely (Fig 8). In such instances the biceps tendon is no longer securely seated within the sulcus or groove but slips out of its normal anatomic site and occupies a position on the inner aspect of the remains of the lesser tuberosity; it is prevented from further displacement posteriorly by a sling formed by the tendon of the subscapularis tendon. In 10 specimens the authors noted that the intracapsular portion of the tendon was absent, and the extracapsular portion had attained a bony anchorage on the shaft of the humerus immediately below the lesser tuberosity.

CLINICAL FEATURES

A distinction must be made between bicipital tenosynovitis encountered in the second and the third decades of life and that encountered after the age of 30 years.

BICIPITAL TENOSYNOVITIS IN THE YOUNG

In this group the basic underlying cause is some congenital abnormality in the bicipital sulcus or groove. The presence of a supratubercular ridge or a marked obliquity of the medial wall of the intertubercular sulcus favors subluxation or even dislocation of the tendon out of the bicipital groove. The author has encountered 7 cases in which palpation disclosed that the tendon slipped in and out of the groove when the arm was abducted and externally rotated, actively or passively.

Generally the syndrome is initiated by an insidious onset, at times it is precipitated by a bout of strenuous activity such as tennis, pitching baseball or shoveling snow and in rare instances it is observed following a fracture of the upper end of the humerus which crosses the bicipital groove. At first the pain is localized over the anterior and medial aspect of the shoulder. Frequently it radiates to the belly of the biceps muscle and even to the flexor surface of the forearm. Occasionally it is projected to the insertion of the deltoid muscle, the inferior angle of the scapula and even to the base of the neck. Not infrequently attention is first focused on the disorder when the patient attempts to touch the posterior surface of the head or the trunk. These movements force the humeral head to glide on the tendon for a greater distance than during other movements of the arm. In the beginning the pain is relieved by rest but is accentuated by activity; later the pain is more or less constant and interferes with sleep. At this time the disorder is commonly and erroneously diagnosed as a bursitis or neuritis. The pertinent physical finding is exquisite tenderness over the intertubercular sulcus. Invariably the examiner can elicit this sign when firm pressure is made over the sulcus. Also rolling

the biceps tendon under the examiner's thumb will produce excruciating pain in all instances. As one would anticipate roentgenologic studies provide no information relative to the nature of the malady.

The clinical course that the disorder pursues depends on the basic underlying factors and the nature of the precipitating agent. In young individuals without a history of trauma, the disease is characterized by recurrent attacks of pain the symptoms are rarely of sufficient severity to cause serious dysfunction. Rest and voluntary elimination of the painful arcs of motion effect a cure in most instances. However if the arm is used excessively recurrent episodes are prone to occur. Only occasionally does the disorder become protracted, increasing steadily in intensity. This is especially true in the small number of cases in which abduction and external rotation of the arm force the tendon out of the intertubercular sulcus or when severe violence is the responsible factor for displacement of the tendon. It has been the author's experience that in this small group of cases conservative measures were of no avail in each instance it was necessary to obliterate the tendon-tendon sheath mechanism by anchoring the tendon to the shaft of the humerus or to the coracoid process in order to effect a cure. It is significant that a frozen shoulder did not develop in any case in the younger age group.

BICAPITAL TENOSYNOVITIS AFTER THE AGE OF 30 YEARS

The onset of the syndrome in this age group does not differ from that described for the patients in the younger age period. However as a rule it is more protracted and more severe not infrequently both shoulders are affected. In this study there were 14 cases of bilateral involvement. As recorded previously the authors are of the opinion that the degenerative alterations in the biceps tendon, usually present during this period of life are responsible for the severity of the symptoms and the protracted

course of the malady. The pertinent and distinguishing feature between the two groups is the development of a frozen shoulder in a large number of cases in the older age period. 38.8 per cent of the cases in this series disclosed varying degrees of frozen shoulder. In these instances a study of the histories revealed that in 80 per cent of the cases bicapital tenosynovitis was the initiating factor which first produced pain, together with varying degrees of restriction of motion of the glenohumeral joint. The arcs of painless motion decreased progressively until the classic clinical picture of frozen shoulder evolved. At this point the authors wish again to point out that any factor which produces muscular inactivity in the region of the shoulder may precipitate the pathologic processes which culminate in a frozen shoulder. Bicapital tenosynovitis is only one of the many causative agents. However once a frozen shoulder has developed, implication of the biceps tendon by the diffuse inflammatory process involving all the soft tissues about the glenohumeral joint is a constant finding.

Pain in the shoulder and limitation of motion of the scapulohumeral joint is not uncommonly noted after fractures and fracture dislocations of the upper end of the humerus. Many observers are of the opinion that the symptoms and the dysfunction are the result of a tendinitis implicating the rotator cuff, partial tears of the cuff or a bursitis. That the aforementioned factors may be the responsible causes in some cases cannot be denied. However the most frequent cause is bicapital tenosynovitis. In fractures or fracture dislocations of the upper end of the humerus the normal configurations of the bicapital groove may be altered seriously or even obliterated. If during the healing process the biceps tendon achieves a bony anchorage on the shaft of the humerus no pain will ensue. On the other hand, if bony anchorage of the tendon fails to occur and if the normal architecture of the groove is sufficiently altered to disturb the normal gliding mechanism of the tendon within the groove

then the frictional forces, acting on the tendon by the altered mechanics in this region, will invariably produce an inflammatory process of the biceps tendon, which gives rise to the painful syndrome. Although no cases of fracture or fracture dislocation of the humerus are included in this series, the authors have encountered a sufficient number of cases of bicipital tenosynovitis and frozen shoulders following the above injuries to justify the formulation of a definitive plan of treatment for such lesions. In the event that surgical intervention is employed in the treatment of these traumatic lesions, the bicipital groove and the tendon are inspected carefully. If the tendon is found to be lacerated or displaced from its normal anatomic position, or if the fracture line traverses the bicipital groove, the tendon is anchored to the shaft of the humerus. By so doing, the gliding mechanism of the tendon is obliterated; this ensures against the development of bicipital tenosynovitis after healing of the fracture has been achieved. Moreover, the absence of pain permits early mobilization of the shoulder. It is significant that in individuals under the age of 30 no instance of bicipital tenosynovitis was complicated by a frozen shoulder, regardless of the severity of the disruption of the upper end of the humerus. This is in striking contrast with the high incidence of frozen shoulders which is encountered past 40 years with or without trauma, particularly between the years of 45 and 55. This observation again emphasizes the premise that in the older patient some "X" factor must be present which predisposes him to the development of a frozen shoulder. As pointed out previously, this "X" factor is the degenerative alteration resulting from physiologic wear and tear and aging.

TREATMENT

Essentially the treatment of bicipital tenosynovitis is conservative regardless of the stage and the causative agent responsible for the disorder. This also includes those cases which exhibit a frozen shoulder in varying



FIG. 9. Roentgenogram showing a staple in position following fixation of the biceps tendon.

stages of development. Rest of the part and restriction of motions within painless arcs will relieve pain and terminate the syndrome in most instances. Irradiation and cortisone given by mouth have been tried with no noticeable effect on the disease. However, hydrocortisone (1 cc.) injected directly into the tendon under the transverse humeral ligament has been a valuable adjunct to conservative therapy. Within the past 10 or 12 months hydrocortisone has been employed in 18 cases. In 10 of these cases the medication produced an immediate beneficial response and definitely shortened the course of the disease. The most dramatic results were noted in those cases which disclosed no evidence of a frozen shoulder. When the latter complication was present the effectiveness of hydrocortisone was less marked and in 4 cases did not alter the progress of the disease. As a rule, 3 or 4 injections of 1 cc. of the solution placed directly into the tendon under the transverse humeral ligament at weekly intervals will produce the desired results. By the end of this period the effectiveness of the medication can be determined.

Surgical intervention was employed in

shoulders. This comprises obliteration of the tendon-gliding apparatus by anchoring the tendon to the coracoid process or to the shaft of the humerus the results obtained are shown in Table 2. In most of the cases in this series treated surgically the biceps tendon was reattached to the coracoid proc-

ess recently this procedure has been modified. Now the tendon is stapled to the shaft of the humerus employing a 3-pronged staple (Fig. 9). This method provides firm fixation of the tendon eliminates much dissection on the anterior aspect of the joint necessary to expose the coracoid process

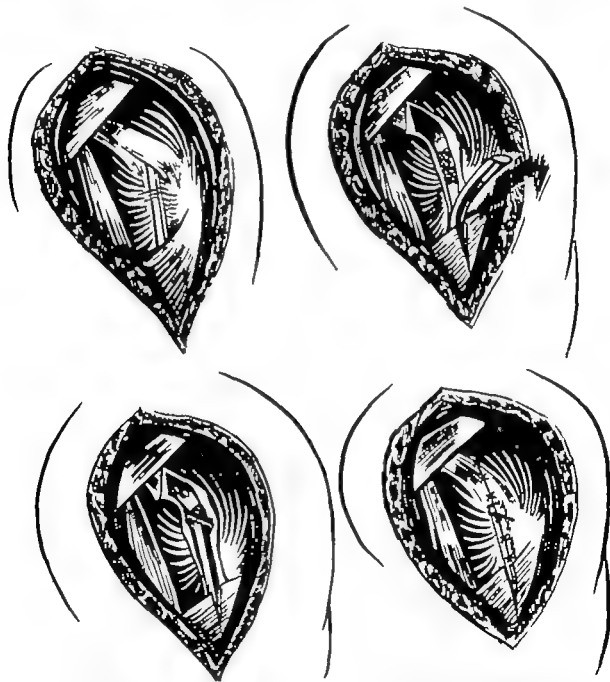


FIG. 10 Steps in the procedure designed to explore the biceps tendon and to anchor it in the bicipital groove (Top left) The biceps tendon is exposed through an anterior deltoid splitting incision (top right) the biceps tendon is severed at its insertion on the supraglenoid bony rim and withdrawn from the joint cavity the proximal end of the bicipital groove is roughened (bottom left) the biceps tendon is stapled to the shaft of the humerus (bottom right) the edges of the musculotendinous cuff are approximated by interrupted sutures.

and converts the procedure from a technically difficult task to a simple operation

OPERATIVE PROCEDURES

The anterior aspect of the shoulder is exposed by a lazy-S skin incision from $3\frac{1}{2}$ to 4 inches long, as described by Hitchcock and Bechtol. After splitting the anterior fibers of the deltoid muscle the biceps tendon and the transverse humeral ligament are identified. The latter is incised longitudinally thereby exposing the extracapsular portion of the biceps tendon. This incision is continued proximally through the fibers of the coracohumeral ligament for a distance of 1 to 2 inches. Now adequate visualization of the inside of the glenohumeral joint is possible. By pulling downward on the arm the biceps tendon is seen as far as its attachment to the apex of the glenoid cavity. At this point the tendon is divided and withdrawn from the joint cavity. Next, the floor of the bicipital groove immediately below the tuberosities is scarified for $\frac{1}{2}$ to $\frac{3}{4}$ inch. The tendon is brought into contact with the raw area and stapled to the humerus by a staple with 3 sharp prongs. This firmly fixes the tendon to its new bed. The excess proximal portion of the tendon is cut off and discarded. The wound is closed in the usual manner.

POSTOPERATIVE MANAGEMENT

After operation no fixation of the arm other than a sling is employed. Usually this is discarded at the end of 5 to 7 days. Motion of the arm below the horizontal (pendulum motion) is started the next day and is performed a prescribed number of times every hour on the hour by the clock. The range of motion increases progressively but always it is maintained within the patient's tolerance. In uncomplicated cases of tenosynovitis optimum restoration of function was achieved in 6 to 12 weeks. In cases complicated by frozen shoulder the period of recovery was longer. In the latter group it must be emphasized that obliteration of

the gliding mechanism of the biceps tendon does not increase the range of motion, but it does relieve the pain. When pain is alleviated or diminished markedly active movements of the arm are possible. This restored muscular activity eventually reverses the pathologic processes producing the diffuse inflammatory disorder characteristic of a frozen shoulder.

ANALYSIS OF CASES REVIEWED

A survey of the cases of bicipital tenosynovitis encountered between January 1948 to June 1953 has been made. This includes all cases of uncomplicated tenosynovitis and those associated with a frozen shoulder. Cases of calcareous tendinitis were not included in this study also cases resulting from fracture or fracture dislocation of the upper end of the humerus were eliminated. There were 77 males and 98 females. The right arm was implicated in 110 and the left in 79. The youngest patient was 16 and the oldest was 69 years of age. The highest incident was between 45 and 55 years (46 per cent) inclusive. Both shoulders were involved in 14 cases. Both shoulders were operated on in 5 cases. Of 175 cases (189 shoulders) in 86 the biceps was transferred either to the coracoid process or to the shaft of the humerus. These cases were subjected to surgery because conservative therapy had failed to alleviate pain and to restore function and in the minds of the authors further continuance of these measures was regarded as futile.

Of the 86 cases operated on 59 provided information pertinent to this study. The period following operation was from 5 to 60 months, with an average of 27 months. The duration of symptoms prior to operation ranged from 2 to 48 months. The average being 15 months. Of the 59 cases, 23 were complicated by a frozen shoulder varying in severity. The age of the patients with this complication ranged from 36 to 69 years. Only 3 cases were under 45 years and 7 cases were under 50 years. Seven patients in this group had involvement of both biceps

TABLE 2. BICIPITAL TENOSYNOVITIS—TREATED SURGICALLY

Case	Age	Sex	Brougher		Dura Time in Months	Compli- cation- Protein Excess	Use Lateral	Bi- Lateral	Injury	No Injury	Sub- Loza Tum	Optimum Res- toration of Function and Range of Motion	Remu- nition	Results			Post Opera- tive Months	Patient Satisfied
			Left	Right										Good	Fair	Poor		
A.L.C.	22	M	+	+	4	+	0	+	+	0	0	4 Months	0	+	+	27	+	
J.M.	24	M	+	+	4	+	0	+	+	0	0	—	0	+	+	13	+	
M.B.	24	M	+	+	7	+	0	+	+	0	0	3 Months	0	+	+	40	+	
M.R.	25	M	+	+	12	+	0	+	+	0	0	6 Months	No Relief	+	+	29	+	
R.W.	25	M	+	+	12	+	0	+	+	0	0	10 Days	No Relief	+	+	30	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	—	No Relief	+	+	43	+	
N.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
A.C.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
C.W.	25	M	+	+	12	+	0	+	+	0	0	2 Weeks	No Relief	+	+	43	+	
F.L.	25	M	+	+	12	+	0	+	+									

Wrong diagnosis: cervical spine pathology. Wrong treatment: manipulated with esthesiograph and severe thrombosis of head.

Unable to explain the failure.	Private inducted into Army	Pala returned when
--------------------------------	----------------------------	--------------------

Summary of Table 2

Total number of cases	59
Males	33
Females	26
Age range	
Duration of illness	16 to 60 years
Complicated with frozen shoulder	2 to 46 months
Unilateral involvement	23
Bilateral involvement	62
Illness with history of injury	7
Hormonal imbalance	19
Hormonal imbalance of London	10
Results: Excellent	6
Good	39
Fair	10
Poor	6
Nonoperative period of evaluation	39 cases—64 percent
Number of patients satisfied	10 cases—16 percent
Number of patients not satisfied	6 cases—8 percent
Number of patients not satisfied	6 cases—10 percent
Number of patients not satisfied	5 to 60 months
Number of patients not satisfied	43
Number of patients not satisfied	6

SUMMARY OF TABLE 3

	Total number of cases
Males	21
Females	53
Totals	8
Age range	21 to 62 years
Cases complicated with frozen shoulder	12
Cases complicated with axillary nerve palsy	23
Unilateral involvement	6
Bilateral involvement	2
Patient's with a history of injury	1
Demonstrable subluxation at onset	1
Period of treatment ranged from	1 to 24 months
Cases (1 month)	9 cases—59 percent
Cases (1-3 months)	14 cases—43 percent
Cases (>3 months)	3 cases—9.6 percent
Results: Excellent	6 cases—16 percent
Good	25
Fair	4
Poor	1
Number of patients satisfied	4
Number of patients not satisfied	1
N. comment.	8
Advanced Surgery (all cases rated poor and fair)	0

TABLE 2. BACTERIAL TENOSYNOVITIS—TREATED CONSERVATIVELY

NAME	AGE	SEX	STOOLAGE		DURATION	COMPLICATED WITHE PROGESS SHOOLAGE	UNI LATERAL	BI LATERAL	IMBURY	NON- IMBURY	SUB- LUTATED	PERIOD OF TREATMENT	RESULTS				PATIENTS SATISFIED
			LARRY	HIGEST									EXCELLENT	GOOD	FAIR	POOR	
B C	40	M	+	+	8 Months	++	++	0	+	+	0	1 Month	+	+	+	+	+
A 0	48	M	+	+	10 Months	++	++	0	+	+	0	2 Months	+	+	+	+	+
A 1 H	48	M	+	+	3 Months	++	++	0	+	+	0	1 Month	+	+	+	+	+
I L C	48	M	+	+	12 Months	++	++	0	+	+	0	2 Months	+	+	+	+	+
L C	45	M	+	+	—	++	++	0	+	+	0	2 1/2 Months	+	+	+	+	+
L C	45	M	+	+	3 Weeks	++	++	0	+	+	0	4 Months	+	+	+	+	+
V N	44	M	+	+	3 Months	++	++	0	+	+	0	6 Weeks	+	+	+	+	+
V N	44	M	+	+	7 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	42	M	+	+	7 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	45	M	+	+	8 Months	++	++	0	+	+	0	3 Months	+	+	+	+	+
L C	45	M	+	+	12 Months	++	++	0	+	+	0	1 Month	+	+	+	+	+
L C	45	M	+	+	16 Months	++	++	0	+	+	0	3 Months	+	+	+	+	+
L C	46	M	+	+	34 Months	++	++	0	+	+	0	2 Months	+	+	+	+	+
L C	46	M	+	+	10 Months	++	++	0	+	+	0	2 Months	+	+	+	+	+
L C	46	M	+	+	1 Month	++	++	0	+	+	0	12 Months	+	+	+	+	+
L C	46	M	+	+	12 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	46	M	+	+	6 Months	++	++	0	+	+	0	1 1/2 Months	+	+	+	+	+
L C	46	M	+	+	3 Months	++	++	0	+	+	0	4 Months	+	+	+	+	+
L C	46	M	+	+	12 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	46	M	+	+	12 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	46	M	+	+	20 Months	++	++	0	+	+	0	—	+	+	+	+	+
L C	46	M	+	+	7	++	++	0	+	+	0	8 Months	+	+	+	+	+
L C	46	M	+	+	1	++	++	0	+	+	0	34 Months	+	+	+	+	+
L C	46	M	+	+	8 Months	++	++	0	+	+	0	3 Months	+	+	+	+	+
L C	46	M	+	+	8 Months	++	++	0	+	+	0	—	+	+	+	+	+

Ad bed tendon turgid. ³Motion has increased, but considerable pain present. ⁸Advised surgery at initial evaluation. ⁴Patient refused surgery.

tendons but both tendons were transferred in only 5 cases. A specific history of injury to the shoulder girdle could be elicited in only 19 patients. In the remainder the onset was insidious. In 5 patients the affected tendon could be subluxated out of the sulcus by external rotation of the arm (Table 2).

Clinical evaluation of the 59 cases disclosed that an excellent result was achieved in 38 (or 64+ per cent), a good result in 10 (or 16+ per cent), a fair result in 5 (or 8+ per cent) and a poor result in 6 (or 10+ per cent). It was interesting to note that 80+ per cent of the patients attained an excellent or good result. All 6 patients who were rated as poor results were not satisfied with the operative procedure; all others expressed satisfaction.

ANALYSIS OF POOR RESULTS

Analysis of the poor results in this group (59 cases) revealed the following information (Table 2). In case A. R., female, 52 years of age, re-examination of the patient disclosed that a wrong diagnosis had been made. In spite of the operation she still complained of severe pain in the shoulder area which was associated with advanced osteoarthritis of the cervical spine. It becomes apparent that lesions of this cervical region must be eliminated before a final diagnosis of bicipital tenosynovitis is made. Brachialgias may be distressing concomitant lesions of the cervical spine. In case C. K., female, age 53 years old, severe pain and limitation of motion was demonstrable immediately after the operation and persisted for 52 months. Careful examination of this patient revealed a serious error in the surgical technique. The biceps tendon stretched from the upper end of the humerus to the coracoid process like a bowstring against which the head of the humerus impinged on external and internal rotation of the arm. It became apparent that the operator had failed to tack the proximal 3 or 4 inches of the tendon of the long head of the biceps brachii muscle to the short head. In two other cases we are unable to determine the cause of

failure. In the fifth case the patient was operated on at the age of 16. He had no complaints up to the time of this survey. 26 months after operation he was inducted into the Army. He was able to do his work, but he did complain (by letter) that he had severe pain in the operative area. The last case G. R., male, aged 60, still complained bitterly of pain 15 months after operation. Examination revealed that the patient had developed causalgia of the affected extremity. The hand was stiff, painful, glossy and of no functional value. In addition, he had severe pain in the cervical spine and over the affected shoulder. Here again advanced osteoarthritis of the cervical spine was noted. However it was difficult to decide whether the causalgia was associated with the cervical spinal pathology or was initiated by the operative procedure.

Table 3 reveals the data on the cases treated by conservative measures. This group also includes some patients who failed to respond to these methods and were proffered surgical intervention, but as yet surgery had not been executed. In this group there were 49 patients ranging in ages from 21 to 64 years; of these 31 cases were reviewed for this study. The remainder failed to answer our inquiries. Twelve of the 31 cases were complicated by a frozen shoulder. 5 cases had bilateral involvement; there were 8 males and 23 females. Subluxation of the tendon was noted in one case.

Evaluation of this method disclosed that even many months after cessation of therapy most individuals still had discomfort which varied in degree. In 7 the pain was severe, and the patients were offered surgical relief. The period of active treatment was from 1 to 38 months, the average being 7 months. The period of time between the institution of the treatment and the making of this survey was 4 to 62 months, the average being 25 months. Excellent results were attained in 29+ per cent, good in 45+ per cent, fair in 9+ per cent, and poor in 16+ per cent. Nevertheless all but 5 of these patients were satisfied with the relief obtained.

Contact was made with 11 of the 45 patients seen for one examination and advised to accept surgical intervention. These were continued on conservative therapy. It was interesting to note that 2 of these were completely free of pain. The periods of time required to achieve these results were 18 and 33 months. One in this group of 11 cases, is reconsidering surgery the others (8 cases) are not improved but still refuse any surgical procedures. The average time since transference of the tendon was first recommended in these 8 cases is 28 months ranging from 4 to 47 months.

CONCLUSIONS

1 Bicipital tenosynovitis is a common lesion causing pain and stiffness in the shoulder joint.

2 The predisposing factors comprise anatomic peculiarities of the region, abnormal functional demands, anomalies of the bicipital groove and degenerative alterations incident to wear tear and aging.

3 The lesion is not associated with a frozen shoulder in patients under 30 years of age.

4 Many cases respond to conservative measures.

5 Surgical intervention is indicated in resistant cases. This consists of obliteration of the tendon-tendon sheath mechanism.

6 A new method of anchoring the biceps tendon has been described.

REFERENCES

- Codman, E. A. *The Shoulder* Boston Private Printing, 1934
- DePalma, A. F. *Surgery of the Shoulder* Philadelphia, Lippincott, 1950

- Loss of scapulohumeral motion (frozen shoulder) *Ann. Surg.* 135 193 1952.
- Frozen shoulder *Am. Acad. Orthop. Surgeons Instructional Course Lectures* 11 313 1952.
- DePalma, A. F. Callery G. E. and Bennett, G. A. Variational anatomy and degenerative lesions of the shoulder joint, *Am. Acad. Orthop. Surgeons Instructional Course Lectures* 6 255 1949
- DePalma, A. F., White J. B., and Callery G. E. Degenerative lesions of the shoulder joint at various age groups which are compatible with good function *Am. Acad. Orthop. Surgeons Instructional Course Lectures* 7 168 1950
- Hitchcock, H. H. and Bechtol, C. O. Painful shoulder: observations of the role of the tendon of the long head of the biceps brachii in its causation, *J. Bone & Joint Surg.* 30-A, 263 1948
- Lippmann, R. K. Frozen shoulder: periarthritis and bicipital tenosynovitis *Arch. Surg.* 47 283 1943
- Meyer A. W. Unrecognized occupational destruction of the tendon of the long head of the biceps brachii, *Arch. Surg.* 2:130 1921
- Spontaneous dislocation of the long head of biceps brachii, *Arch. Surg.* 13 109 1926
- Chronic functional lesions of the shoulder *Arch. Surg.* 35 646 1937
- Spontaneous dislocation and destruction of the tendon of long head of biceps brachii, *Arch. Surg.* 17 493 1928
- Unrecognized occupational destruction of tendon of long head of biceps brachii, *Arch. Surg.* 2:130 1939
- Pasteur F. La teno-bursite bicipitale, *J. radiol. et électrol.* 16 419 1932.
- Schrager V. L. Tenosynovitis of the long head of the biceps humeri, *Surg. Gynec. & Obst.* 66 785 1938
- Tarssy J. M. Bicipital syndromes and their treatment, *New York State J. Med.* 46 996 1946

Recurrent Anterior Dislocation of the Shoulder Joint

JOHN J. GARTLAND M.D.,* AND
JOHN J. DOWLING M.D.*

During recent years some of the confusion surrounding the problem of the recurrent dislocating shoulder has been unraveled by the recorded observations of many competent workers. Thinking had been influenced for years by the teaching of Bankart that ordinary traumatic dislocation and recurrent dislocation of the shoulder were two entirely different entities each with its own peculiar mode of production and pathologic change. In an impressive study Adams¹ has shown that any type of injury which is known to cause an ordinary traumatic dislocation may also cause recurrent dislocation. The incidence rate of the different mechanisms of injury were shown to be about the same in recurrent and nonrecurrent cases.

Along the same lines McLaughlin,² in a study of 101 consecutive primary anterior dislocations reveals a total recurrence rate of about 50 per cent of all lesions without fracture increasing to 90 per cent in all patients under 20 years of age. He feels that treatment of the primary episode is of little importance but that the site and the nature of the primary pathologic disorder is of foremost importance in determining whether or not recurrences would occur. Therefore present-day thinking refuses to set the shoulder joint apart from the other

joints of the body and ascribe to its recurrent dislocations a particular mode of production. Rather it is united in the concept that it is the pathologic change within the joint occurring at the time of injury that sets the stage for recurrences.

If attention is directed to the pathologic anatomy of recurrent dislocations a number of changes are described, each with its own champion. Bankart maintains that detachment of the glenoid labrum, capsule and periosteum from the lip of the glenoid is present in every case of recurrent dislocation and is the *modus operandi* for the repeated dislocations. However evidence is accumulating, which suggests that while this is a frequent abnormal finding it is by no means the sole cause.^{3,4} Many years ago Hybbinette⁵ described the underlying pathologic process as a capsular injury with the formation of an anterior false joint cavity. Since then DePalma⁶ has shown that this so-called false pocket is in reality the subscapularis recess and its related anatomic variants (Fig. 1). Most workers agree as to the presence of a posterior humeral head defect, but Palmer⁷ goes a step further and maintains that it is the primary lesion and that the soft tissue changes are of secondary importance. It is interesting to note that well over 100 years ago Sir Astley Cooper⁸ described the pathologic lesion of recurrent dislocation as a rupture of the subscapularis tendon. He had the opportunity of dissecting two fresh

* Department of Orthopaedic Surgery Jefferson Medical College, Philadelphia

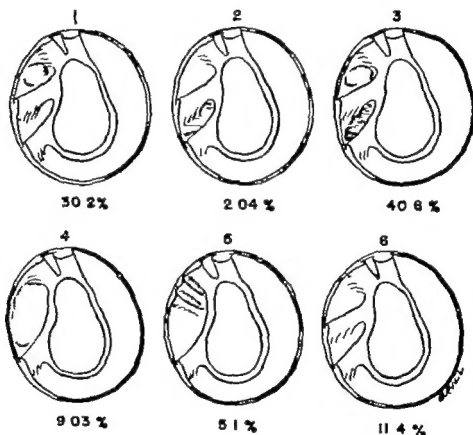
cases of dislocation and found in both that the dislocating head had ruptured the subscapularis tendon in its anterior passage.

When one contemplates the variety of pathologic changes described as the primary lesion of recurrent dislocation it becomes evident that no single constant feature exists. Each of such changes may operate independently to produce a recurrent dislocation. McLaughlin² aptly states the modern concept "the essential pathological condition predisposing to recurrence following traumatic dislocation consists of damage to the antero-inferior supports of the joint, aided and abetted at times by posterior compres-

sion of the humeral head." To this concept DePalma³ adds the theory of neuromuscular imbalance of the short rotator muscles to explain the unique tendency of shoulder joint dislocations to become recurrent dislocations.

TREATMENT

Of the many operative repairs described for this lesion, those that have stood up under the critical evaluation of time and adequate follow up studies have added a buttress to the joint anteriorly or have limited external rotation effectively. The Bankart, the Putti Platt, the anterior bone-block and the Magnuson repairs are all



Types of arrangements of synovial recesses

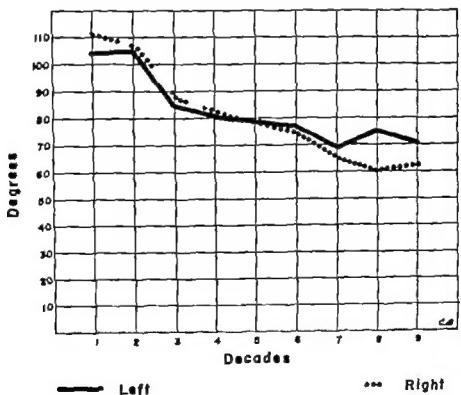
FIG 1 Six types of arrangement of the synovial recesses (1) Characterized by one synovial recess above the middle glenohumeral ligament. (2) One synovial recess below the glenohumeral ligament. (3) Two synovial recesses. A superior subscapular recess above the glenohumeral ligament, and an inferior subscapular recess below the glenohumeral ligament. (4) One large synovial recess above the inferior glenohumeral ligament the middle glenohumeral ligament is absent. (5) The middle ligament exists as two small synovial folds. (6) Complete absence of synovial recesses. (DePalma, A. F. *Surgery of the Shoulder* Philadelphia, Lippincott)

associated with a high degree of success DePalma⁵ has demonstrated that normally the ability to rotate externally decreases with increasing age (Fig. 2) This fact explains the spontaneous cures that occur with aging and is the rationale for those procedures designed to limit external rotation The Magnuson (modified) repair is performed easily and is associated with a comparable degree of success regardless of the underlying pathologic change It is as logical to advocate one type of shoulder repair if it meets the criteria for success as it is to utilize a spinal fusion as definitive treatment of a variety of underlying pathologic changes in the spine It is the purpose of the writers of this report to record the end-results of a series of Magnuson (modified) repairs for recurrent dislocating shoulder joints

OPERATIVE PROCEDURE

As performed in this institution * the repair described by Magnuson⁶ has been modified slightly by transferring the subscapularis tendon not only laterally but also distally on the shaft (Fig. 3) As described by DePalma,⁵ the operation is performed through an S-shaped incision on the anterior aspect of the shoulder joint, beginning at the inferior margin of the acromioclavicular joint. The interval between the deltoid and the pectoralis major muscles is developed, taking care not to injure the cephalic vein, which is retracted medially with the pectoralis major By externally rotating the shaft of the humerus the subscapularis tendon, as it inserts into the lesser tuberosity comes

* Jefferson Medical College Philadelphia.



Average range of external rotation at each decade

FIG. 2. Graph showing gradual decrease in range of external rotation with advancing age. Study was made on 800 individuals, 100 in each decade from the first to the eighth. Only individuals who never suffered a dislocation or who never had severe injuries to the shoulder joints were selected for this investigation. (DePalma, A F Surgery of the Shoulder Philadelphia, Lippincott)

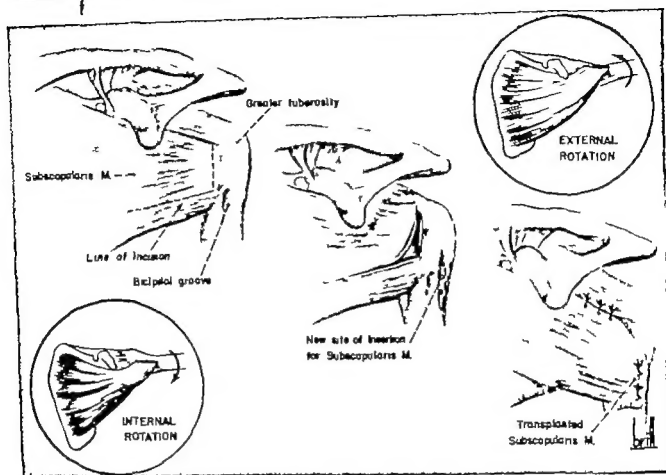


FIG 3 Magnuson operation (modified) for recurrent dislocation. The end of the subscapularis tendon is transferred to a bony trough below the greater tuberosity and parallel to the posterior lip of the bicipital groove (DePalma, A. F. *Surgery of the Shoulder Philadelphia, Lippincott*)

into view. A blunt dissector is passed under the subscapularis tendon in order to determine more clearly its upper and lower borders. An incision is made in the interval between the supraspinatus and the subscapularis muscles, beginning proximally to the blending of the subscapularis tendon with the fibrous capsule. The incision is continued to the anterior lip of the bicipital sulcus. A second incision the same length as the first, is made along the lower border of the subscapularis muscle to the tendon between the two incisions, which is then freed from the anterior lip of the bicipital groove by sharp dissection. Medial retraction of the tendon and the capsule affords a clear view of the humeral head, the anterior glenoidal margin and the anterior portion of the synovial capsule with its glenohumeral ligaments and bursal recesses.

The greater tuberosity is visualized by rotating the arm internally. The subscapularis is then pulled across the bicipital groove by means of a suture through its substance to a point below the greater tuberosity where its site of reattachment is determined. The tendon should be anchored to the humeral shaft below the level of its original insertion under moderate but not severe tension. With a thin osteotome a slot, $\frac{1}{4}$ inch wide and as long as the width of the subscapularis tendon is made parallel with the posterior lip of the bicipital groove below the greater tuberosity. Three or four drill holes are then made in the posterior lip of the newly formed slot, and the end of the tendon is buried in the bony trough with silk mattress sutures. The upper border of the subscapularis muscle is approximated to the supraspinatus muscle by side-to-side su-

tures while its lower border is sutured to the capsular tissues under the head of the humerus. Considerable restriction of external rotation is demonstrable at the completion of the procedure. The subscapularis muscle and the tendon fibers can also be seen forming a sling under the head on abducting the arm in internal and external rotation. The procedure is completed by wound closure in layers with interrupted sutures.

Postoperatively the arm is fixed to the side with the forearm across the chest by means of a plaster Velpeau dressing. This dressing is kept on for a total of 6 weeks. Following removal of the cast gradual resumption of activities and all motions is allowed. As a rule abduction is restricted a few degrees and external rotation may be restricted as much as 50 per cent. This usually produces no functional disability and is assurance against redislocation.

ANALYSIS OF RESULTS

In 1950 DePalma reported the results of 23 recurrent dislocating shoulders treated by the modified Magnuson operation during the

period May 1945, to August, 1948.⁸ The recurrence rate of dislocation in this series was 8.7 per cent.

Complete records are available on 20 additional cases operated on during the period January, 1949 to April, 1953. The end results of these cases form the basis of this report (Table 1). Complete follow-up data are available in 19 of these 20 cases. One patient moved to a distant city and was lost to follow-up. This group comprised 15 males and 5 females, ranging in age from 15 to 37 years. The follow up period ranged from 8 months to 5 years and averaged 2½ years. The recurrence rate of dislocation in this group was 10.5 per cent. Thus the known recurrence rate in the 43 cases available for study between 1945 and 1953 is 9.6 per cent.

SUMMARY

1. It is no longer tenable to set the shoulder joint apart from the other joints of the body and ascribe to its recurrent dislocations a particular mode of production.

2. Damage to the antero-inferior supports of the joint, aided and abetted at times by posterior compression of the humeral head,

TABLE 1

NAME	SEX	AGE	DATE OF OPERATION	NUMBER OF RECURRENCES	PAIN	SATISFIED
A. F.	F	22	7-30-51	0	Yes	Yes
C. M.	M	37	7-24-51	0	No	Yes
P. W.	M	22	9-5-50	0	No	Yes
P. W.	M	22	1-17-51	0	No	Yes
C. F.	M	27	1-20-53	0	No	Yes
W. B.	M	19	3-17-50	3	Yes	No
D. L.	M	17	1-23-49	0	No	Yes
J. M.	M	16	7-28-49	0	No	Yes
P. G.	F	26	7-25-50	No Followup		
F. C.	M	35	8-6-50	0	Yes	Yes
J. C.	M	19	8-25-49	8	Occasional	No
R. M.	F	25	7-12-50	0	Occasional	Not completely
A. M.	M	24	7-13-50	0	Yes	Yes
A. J.	M	24	4-22-53	0	No	Yes
I. S.	M	23	7-14-52	0	No	Yes
J. W.	M	24	1-20-53	0	No	Yes
G. A.	F	17	1-22-51	0	Yes	Yes
A. C.	M	15	5-14-52	0	Occasional	Yes
K. L.	F	31	10-31-51	0	No	Yes
P. H.	M	18	7-30-51	0	No	Yes